# HYDRODYNAMICS OF THE HUMAN CIRCULATORY SYSTEM: A REVIEW

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# NAVAL POSTGRADUATE SCHOOL
## Monterey, California


# THESIS

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September 1972

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Submitted in partial fulfillemnt of the
requirements for the degree of

MASTER OF SCIENCE IN AERONAUTICAL ENGINEERING

from the

NAVAL POSTGRADUATE SCHOOL
September 1972

ABSTRACT

The results of a literature survey of blood flow are used to determine the extent to which the principles of hydrodynamics are being employed in the study of the human circulatory system. Blood flow is categorized by such parameters as Reynold's number, vessel diameter and geometry, and transport mechanism with a view toward understanding the rheological behavior of blood in various flow regimes.

This study provides an engineering explanation of the hydrodynamic origin of such physiological disorders as arteriosclerosis, atheroma, and thromboembolism. Moreover, it provides an appreciation for the engineering aspects in the design of cardiac assist and cardiac replacement devices.

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# I. INTRODUCTION

In an age where engineers and doctors have teamed up in an effort to understand the complexities of the human body, one of the most difficult tasks in this endeavor lies in the analysis of the human circulatory system.[1] Consider the function served by the most prodigious muscle in the human body, the heart. Heart muscle contracts approximately thirty-six million times a year, during which it pumps roughly 657,000 gallons of blood, based on its dormant operation. On the average, it generates enough energy in twelve hours to raise a weight of 65 tons one foot off the ground. It is one of the few muscles which never rest, and the life-span of the human body is contingent upon the ability of the heart to perform its never-ending task.[2] Add to these considerations the myriad of arteries, veins and capillaries which transport the life-giving blood under a variety of flow conditions, and one has all the ingredients of an engineering miracle.

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[1] That this is an age-old interest is demonstrated in Circulation of the Blood: Men and Ideas, a book which spans the gap between present day technology and the crude but remarkably accurate Hippocratic document (circa 4th century B.C.) describing the heart and its valves (Fishman and Richards, The Oxford University Press, New York 1964).

[2] Biology; An Introduction to the Science of Life, Goodnight, Goodnight and Armacost. John Wiley & Sons, Inc., New York, 1962, pg. 61.

Most of us are aware of the tangible evidence of work being done vis-à-vis the human circulatory system, the most prominent of which might be the perfection of the surgical aspect of the heart transplant, first achieved independently by Doctors Christian Barnard and Denton Cooley. In addition, a host of prosthetic devices are being perfected: a ventricular assist device (to ease the burden of an ailing heart), artificial heart valves, and even a completely mechanical heart. Finally, devices such as the pacemaker and the heart-lung machine are in common usage today. What might not be so apparent is the role played by the engineer in the design and adaptation of these devices to the human body. With this in mind let us look at the circulatory system from an engineering viewpoint, inasmuch as the most brilliant surgical techniques and machinery are worthless if they in any way disturb the optimum hydrodynamics of the system.

## II. BLOOD

Before examining the variables of blood flow in the tubing of the human body, it seems prudent to consider the rheological properties of the blood itself. The complexity of the study is at once apparent from the composition of the blood, broadly consisting of plasma and formed elements.[1] Blood plasma occupies 55% of the blood volume and consists, by weight of

| | | |
|---|---|---|
| 1. | water | 90% |
| 2. | inorganic salts | 1% |
| 3. | major proteins | 7% |
| 4. | other substances ($O_2$, $CO_2$, wastes, etc.) | 2% |

Formed elements consist of three major categories, namely erythrocytes (red cells), leucocytes (white cells) and thrombocytes (platelets). The red cells vary in number from four to six million per cubic millimeter of blood, and are the oxygen carriers. The platelets are the clotting agents and vary in number from 200,000 to 800,000 per cubic millimeter of blood. The white cells, averaging 6,000 to 10,000 per cubic millimeter of blood are further classified as:

| | | |
|---|---|---|
| 1. | polymorphonuclear neutrophils (occurring as pus at infection sites) | 50 to 75% |

[1]Unfamiliar terms encountered in this text may be found in a glossary of terms at the end of the paper, as are the original sources of diagrams which are referred to from time to time.

2. polymorphonuclear eosinophils (parasitic and allergic diseases) 2 to 4%
3. polymorphonuclear basophils 0.5%
4. lymphocytes 20 to 40%
5. monocytes 3 to 8%

Initially, a distinction must be made between the Newtonian and non-Newtonian character of blood. A Newtonian fluid is one whose shearing stress is related to the rate of strain in the form:

$$\tau = \mu \frac{du}{dy}$$

where the dynamic viscosity $\mu$ is an intensive thermodynamic property of the fluid, and independent of the fluid motion. In contrast, the viscosity of a non-Newtonian fluid at a given temperature and pressure is a function of the velocity gradient (du/dy). Goodnight, et al, in Biology: An Introduction to the Science of Life, define "hematocrit" as the "percentage by volume, of formed elements to the total volume of blood. . . ." Since the red blood cells compose 97% of the total cell volume (formed elements) in the blood, their removal would render the blood essentially Newtonian in nature. Here then is the significance of the hematocrit in the Newtonian (or non-Newtonian) behavior of blood:

1. Blood of low hematocrit (less than 20%) is essentially Newtonian in behavior.
2. Blood of high hematocrit (up to 67%) and high shear rate is generally Newtonian in behavior.[1]

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[1]This is graphically represented on pg. 76, Biomechanics, Its Foundations and Objectives, Fung, et al.

3. Blood of intermediate hematocrit and lower shear rate is non-Newtonian, and behaves as a Casson Fluid.[1] (See Appendix A).

While it might appear that high hematocrit implies a non-Newtonian character, the dynamic effect of the high shear rate is to produce a "bolus flow" in which the viscosity of blood approaches that of plasma, a Newtonian fluid. In a paper prepared by AVCO Everett Research Laboratory,[2] the authors attribute the non-Newtonian behavior of blood at low shear rates to the propensity of the red cells to form long chains (rouleaux) like poker chips. The adhesion of these cells to one another may be broken at higher shear rates, and therefore blood is what is termed in rheology a pseudoplastic or shear-thinning fluid.

A. C. Burton takes an interesting approach in discussing the design and mechanical function of the blood and its components.[3] The approach taken is one in which the "job requirements" of the red blood cell are enumerated, thereby establishing the criteria for the design of these hemoglobin vehicles. For example, the need for erythrocytes as carriers of hemoglobin arises upon the realization that at the same concentration of free hemoglobin in the plasma, the

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[1]The mathematical model of a Casson Fluid is developed in the Mathematics of Pulsatile Flow in Small Vessels (Rand Corporation) by Aroesty and Gross, discussed in Appendix A.

[2]The Fluid Mechanics of Thrombus Formation, pg. 15.

[3]Circulatory and Respiratory Mass Transport; "The Mechanics of the Red Cell in Relation to Its Carrier Function," A. C. Burton, pp. 67-84.

viscosity of blood flowing through the vessels would be orders of magnitude greater. The net effect would be a requirement for intolerable operating pressures in the circulatory system and, by extension, increased cardiac output.

With regard to size, given a maximum capillary diameter of 10μ, the red blood cell can be as large as 8μ in diameter. The reason for this, while not intuitively obvious, is related to a phenomenon known as the Fahreous-Lindqvist effect which is observed in micropipettes (as capillary models). In the microcirculation, where cells are of the same order of size as the diameters of arterioles, capillaries, and venules, it has been observed that the hematocrit and apparent viscosity of the blood decrease as the tube diameter is reduced. The physical explanation of this Fahreous-Lindqvist phenomenon is found in Whitmore's axial train model.[1] Observations of flow in the capillary beds of the microcirculation has shown that cells tend to follow each other axially in single file with their discoidal surfaces roughly perpendicular to the axis of the vessel. This train of cells and interspersed fluid tends to travel as a single cylindrical unit of infinite viscosity, surrounded by an annulus of suspending fluid (plasma) in which the shearing takes place. The viscous drag of the annulus fluid on the intra-cellular fluid leads to the

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[1] Rheology of the Circulation, Whitmore, pg. 128.

development of "bolus flow" in the axial train model, as depicted in Figure 1. (See also Fig. 8.11 in Rheology of Circulation, Whitmore.) The "bolus flow," a passage of single red cells separated by trapped plasma, uses so little energy that the effective viscosity approaches that of plasma, even at high hematocrits. For this reason, the major part of the total resistance to flow is not in the capillaries (about 25%) but in the arterioles (about 60%).

The shape of the red blood cell is determined by the deformations it must undergo in the capillaries, arteries and veins, the relative sizes of which are seen in Figure 2. The intolerance of the cell membrane to stretch makes it mandatory that the cells be non-spherical, since for a given volume the sphere has less surface area than any other shape into which it might deform. In an article entitled "Theory of the Sphering of Red Blood Cells," authors Y.C.B. Fung and P. Tong develop a rigorous mathematical solution for the sphering process. Based on the biconcave geometrical shape of the red cell and the thinness of the cell wall, the implication is that the cell wall can be deformed into an infinite number of applicable (isometric) surfaces; consequently the red cell is capable of large deformation without inducing membrane stresses. The actual biconcave discoid[1]

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[1]The distinctive biconcave shape of the human erythrocyte is attributed to a balance of forces on the membrane. In an article entitled "On the Shape of Erythrocyte," Lopez, et al provide a numerical study on distribution of these forces, considered to be electrostatic, hydrostatic and tensile (membrane) in nature.

shape of the red cell is sufficiently non-spherical to allow a great deal of deformation, even to the extent of enabling it to pass through tubes 3.7μ in diameter.[1] In fact, this limiting diameter of approximately 3.7μ, occurring in the spleen, is what filters out the old cells (rejecting them as being of inappropriate geometry).

The optimum value of hematocrit is determined by two inversely related characteristics: viscosity and flow rate. L. E. Bayliss[2] points out the certainty that the reduction in the apparent viscosity of blood, as derived from the value of the pressure/flow ratio, is due not only to a progressive increase in width (i.e., decrease in viscosity) of a marginal slippage zone; it must be supposed that the viscosity of the blood itself varies inversely with the shearing stress to which it is subjected. According to the simple "Bingham" conception,[3] it is supposed that when the pressure applied to the ends of a tube containing blood is small, there is a "plug" flow, and an unsheathed core of blood slides as a whole within a slippage zone provided by the wall effect. True plug flow will occur only if each cell is attached to

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[1] In an article entitled "The Physics of Blood Flow in the Capillaries, III. The Pressure Required to Deform Erythrocytes in Acid-Citrate-Dextrose," Prothero and Burton provide experimental evidence of the steady passage of mammalian red cells, at low pressure, through pores of 5.0 or 3.0 microns in diameter, with no evidence of hemolysis.

[2] Flow Properties of Blood, pp. 29-62.

[3] See Rheology of the Circulation, Whitmore, pp. 44 and 45.

several others in such a way that some kind of three-dimensional lattice-work structure is formed: if the relative cell volume is not sufficiently large for this to occur, there might still be some aggregation of cells into clumps; and the change from a few isolated aggregates to an almost solid structure is apt to occur as a result of a very small increase in relative cell volume above some critical value. This critical value then represents the minimum hematocrit. At a higher hematocrit, more hemoglobin is being transported in the blood, but due to the higher associated viscosity, this transport takes place at a relatively slower rate. The calculated optimal value for maximum efficiency of oxygen transport is found to be close to the normal hematocrit for the species. Examples are given on page 74, Figure 6 in Circulatory and Respiratory Mass Transport, and for man, normal hematocrit turns out to be 47%.

Much experimental evidence has been compiled to show that while human blood is a non-Newtonian fluid, plasma behaves as a Newtonian fluid. Hence, depending on the Reynold's number, viscosity, hematocrit and shear rate, the total spectrum of blood flow in the human body can be mathematically modeled, extending from the "bolus" laminar flow in capillaries and large arterial flow, to the turbulent flow regime in the heart during systolic ejection. The rheological behavior of blood can vary greatly throughout the microcirculatory bed. In the larger arterioles,

blood may be considered a continuum and there appears to be a general agreement that its rheological behavior can be modeled by a Casson fluid (see Appendix A) which has both finite yield stress and shear-dependent viscosity. For smaller arterioles ($D < 100\mu$), the red blood cells appear large enough so that the continuum assumption is no longer valid. In the smallest capillaries, the red blood cells have the same magnitude of dimension as the microvessels and the bolus flow model applies, with red blood cells moving single file through the vessel separated by packets or boluses of plasma.[1]

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[1] "The Fluid Mechanics of Pulsatile Flow in the Microcirculation," Aroesty and Gross, pp. 2-3.

## III. PASSAGE STRUCTURE

Having examined the rheological properties of blood, "the fluid," the next logical step is to consider the structure of "the piping" in which this fluid must flow. The blood vessels in the human circulatory system are defined in relation to the heart: arteries lead away from the heart; veins toward the heart; and capillary networks connect the arteries with the veins (see Fig. 3).

The heart wall is one of almost pure muscle covered on the outer and inner surface with slippery endothelium (smooth, flat cells arranged more or less like the stones of a pavement). This endothelial layer is also common to arteries and veins, and its design is such as to offer minimum resistance to flow through the vessels.[1] The outer endothelial layer (pericadium) permits the heart to slip to and fro in the chest cavity while the inner endothelial layer (endocardium) is arranged in pleat-like folds, forming four sets of valves (see Fig. 4). The atrioventricular valves prevent backflow of blood from the ventricles to the atria during systole, and the Semilunar valves prevent backflow from the aorta and pulmonary arteries into the ventricle during diastole. All these valves open and close passively, i.e., closing when a backward pressure gradient

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[1] Principles of Modern Biology, Marsland, pp. 278-279.

pushes the blood backward and opening when a forward pressure gradient forces the blood forward. The construction of the different valves is in keeping with the dynamic stresses to which they are exposed: the A-V valves, subjected to higher pressure gradients and ejection velocities than found in the semi-lunar valves, require the support of a papillary muscle network. Moreover, the snapback of these mitral (tricuspid) valves gives rise to a much greater mechanical abrasion than found in the aortic valves, a fact of considerable importance in artificial valve design.[1] These valves separate the heart into four chambers: two pumping stations (right and left ventricles) and two receiving stations (right and left atria). The mechanics of the heart as a high pressure pump, together with the types of fluid flow this pumping entails, will be discussed in Section IV.

The structural similarity of arteries and veins makes it possible to describe them in essentially identical terms, with some notable exceptions and variations. The same three layers of tissue are present in the walls of the artery and vein, although these layers are thicker and more clearly defined in the artery (see Fig. 2). The inner layer is the endothelium. The middle layer is a sheath of visceral muscle, flanked both inside and out by elastic connective tissue; and the outer layer is composed of loose connective tissue. The fibers of the muscle sheath tend to encircle

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[1] Textbook of Medical Physiology, Guyton, pp. 153, 154.

the artery, permitting it to change its caliber according to the needs of the tissue. This variable geometry of the arteries, (discussed later in the Windkessel model of arterial flow) enables the arteries to maintain the blood pressure during the ventricular diastole (rest period), assuring a continuous flow in the capillary system.[1] A distinctive feature of the venous structure is that most veins possess valves resembling those of the heart, but more flimsy in nature (see Fig. 5); only those large veins running up from the trunk to the heart and from the head down to the heart lack them. Since blood enters the veins at a relatively low pressure and must be lifted against gravity, valves are required to prevent the weight of blood above a point from impeding the circulation up to that point. That these valves work in conjunction with large skeletal muscles to produce venous flow will be shown in Section IV.[2]

As might be expected of the smallest vessel in the circulatory system, the capillary consists merely of a naked wall of endothelium. The diameter of some capillaries is so small that blood corpuscles must pass through in single file, and several hours may be required for a single milliliter of blood to traverse its length. The various flow regimes in the microcirculation previously mentioned in the

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[1] Principles of Modern Biology, pp. 279-280

[2] The Body Functions, pp. 135-136.

discussion of the rheology of blood, will be covered in more detail in Sections IV and V.

## IV. TRANSPORT MECHANISM

Before considering the flow regimes of the circulatory system, it is advisable to examine the transport mechanism which is predominant in different portions of the body. Again, the heart is the most logical starting point, since it is the primary driving force. As stated earlier, the four chambers of the human heart constitute the two receiving stations and two pumps which account for the pumping mechanism of the heart. The heartbeat is a synchronous muscular contraction of different areas of the heart stimulated by the heart's pacemaker, the sinatrial (SA) node and conducted to the Purkinje fibers via the Atrioventricular bundle. The electrically generated signal leaves the SA node and arrives at the AV node some 0.04 seconds later. This delay, together with that in transmitting the signal from the AV node to the AV bundle, allows sufficient time for atrial contraction prior to the commencement of ventricular contraction. The Purkinje fibers, after originating in the AV node, form the AV bundle and terminate in the cardiac muscle fiber of the ventricles where they stimulate ventricular contraction. The timing of the cardiac impulse is depicted in Figure 6. In this manner, the heart exhibits pulsation, a rhythmic activity in which a nearly synchronous contraction, the systole, alternates with a period of relaxation and filling, the diastole. Based on a heartbeat

of 70 per minute, the ventrical systole is roughly 0.3 seconds while the atrial systole is 0.1 seconds. The diastoles are longer by comparison, the atrial diastole being 0.7 seconds and the ventricular diastole being 0.5 seconds. A single pulsation, the cardiac cycle, consists of the heartbeat itself and a refractory period during which the heart cannot be stimulated to beat and consequently is allowed to rest. The atrial and ventricular systoles take up only half the cycle and the heart relaxes and fills in the other half.[1] A schematic of the pressure pulses throughout the cardiac cycle is presented in Figure 7. With regard to the mechanism of this pumping action, it should be pointed out that blood moves into the ventricles from the atria apparently because of the elastic recoil from the contraction of the ventricles. The atria contract, but this only transfers a small amount of remaining blood into the ventricles.

In his article in International Symposium on Pulsatile Blood Flow (pp. 1-14), E. O. Attinger points up a key distinction in the musculature of the heart. The thick-walled, cylindrically-shaped left ventricle, characterized by an essentially concentric constriction pattern, is architecturally designed as a high pressure pump. (Figure 8 points out the major portion of the pressure load assumed by the left ventricle.) In contrast the surface area of

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[1]Biosphere: A Study of Life, p. 635.

the thin-walled right ventricle is large compared to the volume of the cavity, and the wall contracts with a minimum of myocardial shortening. The efficiency of the heart (i.e., ratio of actual work performed to total energy consumed) is in the order of 20 to 25%. At rest, a man's heart takes up about 5% of the cardiac output and accounts for about 10% of the total oxygen consumption. In heavy exercise, however, the heart alone may consume as much oxygen as does the whole body at rest. Since the body operates at the same low level of mechanical efficiency, the heart is required to expend an appreciable amount of energy in supporting the performance of a physical work load. From this point of view, therefore, anything which contributes to the efficiency of the cardiovascular system is of extreme value to the animal.

Each time the left ventricle contracts, it empties its full blood contents into the arterial system. Since this surge of blood cannot flow simultaneously through the fine capillary network, the arteries must stretch. Then, in the short interval between heart beats, the elastic recoil of the stretched arterial walls tends to maintain blood pressure, assuming continuous flow in the capillary system. M. G. Taylor discusses the peristaltic mechanics of pulsatile flow in the arterial system[1] as follows: It is known that in the arterial system the distensibility is variable from artery

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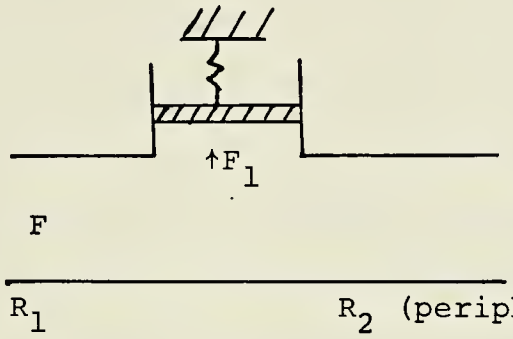
[1] Circulatory and Respiratory Mass Transport, "The Optimum Elastic Properties of the Arteries," pp. 136-152.

to artery and shows a decrease in the more peripheral arteries; it has also been observed that all arteries show a progressively decreasing distensibility as they are inflated. When one speculates upon the possible physiological significances of this non-linearity, it appears to confer two useful properties upon the system. First, thinking of the artery as a Windkessel capable of storing




where $R_2 > R_1$

potential energy, if the arteries become progressively stiffer as they are further distended, there is a reduction in the amount of work which the heart must perform simply to inflate the Windkessel. Second, an increasing stiffness of the Windkessel leads to a reduction in the time required for the pressure to rise after an increase in the peripheral resistance. This second benefit would effect an increase in the speed of response to reflex adjustments of the peripheral resistance, given a constant cardiac output. In Continuum Mechanics in Biomedical Systems, Y. C. Fung offers the possibility of a peristaltic mechanism in the circulation of blood, citing the fact that the arterioles and venules in the bat's wing have been observed to vary their diameters periodically.

Due to substantial pressure head losses in the arterioles, capillary flow occurs at extremely low Reynold's numbers (i.e., $Re \ll 1$). The transport mechanism is still a pump effect, with some assistance from osmotic gradients as peripheral tissue and organs lure the blood in for exchange of absorbed gases, proteins, etc. In terms of the "bolus flow" model which pertains to capillary flow, the amount of plasma leaking back past the red blood cell is small compared with the total flow. As a result, most of the fluid in the bolus must perform a circulating motion, relative to the red cells, to ensure that the mean plasma velocity be almost equal the mean cell velocity. Such a flow may convectively assist the mass transport of slowly diffusing nutrients stored in the plasma, such as fats and proteins.[1] The transport role of plasma in the bolus flow model is given further credibility in a paper by Prothero and Burton entitled "The Physics of Blood Flow in Capillaries, I. The Nature of Motion." These authors gathered experimental proof that gaseous equilibrium was considerably accelerated by bolus flow (as observed in pulmonary capillaries), and that bolus flow was up to twice as effective in transferring heat as was Poiseuille flow. There is even evidence of

[1] "Plasma Motions in Narrow Capillary Flow," J. M. Fitzgerald, Journal of Fluid Mechanics (1972), vol. 51, part 3, pp. 463-476.

pulsatile flow in the capillary network (microcirculation)[1] and the concept of a "lubricating" cell-free plasma layer becomes a key element in the transport mechanism.

As blood filters out of the capillary network into venules, at low velocity, the system is faced with the problem of returning this blood to the heart, against gravity. This transport would require considerably more pressure than is available to move against a column of blood four feet high. To circumvent this problem, blood is raised in the veins by stages, the low pressure being augmented by contracting skeletal muscles in which the veins are embedded. The staging is accomplished by sequenced valves which open and close to sustain the compartmented blood as it is "milked" upward. This mechanism is often referred to as the "venous pump," and by virtue of its staging offers a peristaltic transport mechanism. The venous valves are shown in Figure 5. Once the blood nears the chest, changes in intrathoracic pressure caused by breathing, help suck blood into thoracic veins, from which blood is drawn into the heart by the pressure reduction created each time the heart empties itself.[2] The pressure variations throughout the circulatory system are shown in Figure 9.

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[1] See Aroesty & Gross, "The Fluid Mechanics of Pulsatile Flow in the Microcirculation," See also, M.J. Lighthill's "Pressure Forcing of Tightly Fitting Pellets Along Fluid-filled Elastic Tubes," Journal of Fluid Mechanics (1968), Vol. 34, part 1, pp. 113-143. Lighthill discusses the degree of lubrication in vessels of different size.

[2] Biosphere: A Study of Life, p. 644.

These are basically then the transport mechanisms used to carry the blood through the human body under normal conditions. Extensive research has been done on the effects of increased gravitational acceleration and increased pressure on these mechanisms,[1] but these are extremum applications.

Having considered the mechanism provided by the human body to circulate blood, it remains to consider the mechanics of transport through the cell membranes and into the body tissue. Substances are transported through the cell membrane by two major processes, diffusion and active transport. Diffusion means the movement of substances in a random fashion caused by the normal kinetic motion of matter, whereas active transport means movement of substances as a result of chemical processes that impart energy to cause the movement. A. C. Guyton discusses the factors affecting the diffusion rate of a substance from one area to another:

1. The greater the concentration difference, the greater is the rate of diffusion, (when the substance is water, the diffusion is called osmosis).
2. The less the molecular weight, the greater the diffusion rate.

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[1]For example, see AGARD Conference Proceedings NO 65 (AD 711980):
a) "The Role of Hemodynamics in Transport of Inert Gases in the Peripheral Circulation," by Lt R. G. Buckles and
b) "Gravity Dependence of Pulmonary Capillary Blood Flow," by D. H. Glaister.

3. The shorter the distance, the greater is the rate.
4. The greater the cross section of the chamber in which the diffusion is taking place, the greater is the rate.
5. The greater the temperature, the greater is the molecular motion, hence the greater is the rate of diffusion.[1]

Diffusion may be either free (in case of substances readily soluble in the lipid matrix of the cell membrane, such as oxygen and water) or facilitated (by carrier substances enabling membrane passage by substances normally insoluble in the lipid matrix, such as sugars). When diffusion must occur against a concentration gradient (active transport), a process similar to facilitated diffusion occurs but little is known about the mechanisms by which energy is utilized in the transport mechanism.

Without going into great detail then, it suffices to say that the red blood cells provide the packaging for the sugars and the proteins which nourish the body tissue. By means of an ingeniously designed body chemistry, these substances are exchanged between inter-and extra-cellular fluids, and by the same processes of concentration difference, electrical potential difference and pressure difference are transported into the body tissue.

[1]Guyton, Textbook of Medical Physiology, p. 40.

## V. BLOOD FLOW

Generally speaking, blood flow may be broadly described as being steady or pulsatile, laminar or turbulent. Moreover, depending on the hematocrit and shear rate, the blood itself may be Newtonian or non-Newtonian. In view of these facts, there are many different flow situations in the human circulatory system. R. L. Whitmore[1] postulates a general conclusion, drawn from "in vitro" experiments and a knowledge of the dimensions and velocity rates in living bodies, that the flow in all but the largest arteries is too slow for turbulence to occur (Bayliss, 1952; Scott Blair, 1958).[2] Moreover, at low speeds of flow, rouleaux formations (stacking of red blood cells) may cause the blood to become non-Newtonian in behavior, but during most flow measurements the velocity is sufficient for this effect to be unimportant. Hence, in order to develop a good approximation of blood flow in narrow tubes (capillaries), it is only necessary to assume that blood, in bulk, behaves as a simple Newtonian fluid.

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[1] International Symposium on Pulsatile Blood Flow, pp. 63-83.

[2] See Rheology of the Circulation, Whitmore:
Fig. 7.1, pg. 91 - Plot of Pressures and Pressure Variations at different locations in Circulatory System.
Fig. 7.3, pg. 93 - Vessel Size, Flow Velocity and Re for Different Vessels in Human Circulatory System.

Some apparently contradictory yardsticks have been established for the determination as to whether flow is laminar or turbulent. For steady flow conditions in pipes, Reynolds (1883) showed that the "streamline" flow of dye filaments is only permanently broken up when the Reynold's number exceeded a "lower critical value," normally 2000. Reynolds number is defined as:

$$Re = \frac{vd_c\rho}{\mu}$$

where v is the man velocity of flow, $d_c$ is the internal diameter, $\rho$ is the density and $\mu$ is the viscosity. R. L. Whitmore cites a Reynold's number of 2300 as this lower critical value, above which a certain disturbance is necessary to initiate a stable turbulence, and the larger the Reynold's number, the smaller need be the initial disturbance.[1]

The yardstick falls short in its application to blood for a variety of reasons:

1. In models with sharp bends and various bifurcations, the streamline pattern of dye filaments is broken up at Reynold's numbers much lower than 2000. As to whether or not this constitutes turbulent flow, one would have to examine the downstream characteristics, inasmuch as large ring vortices at branch sites could cause a purely local disturbance of flow pattern, hence one which is not strictly turbulent.

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[1] Rheology of the Circulation, R. L. Whitmore, p. 39.

2. The fact that blood flow is pulsatile in nature introduces a variation in Reynold's number with time. For example, Katz and his colleagues [(1944) American Journal of Physiology. 159,483] showed that in the human ascending aorta, the Reynold's number, calculated from the mean systolic velocity, ranged from 12,000 to 20,000. Since the peak velocity will be at least twice this value, Reynolds numbers of the order of 40,000 are possible, and flow is clearly turbulent. At other periods of the cycle, the Reynold's number will fall to zero, and throughout the diastole it will be low.[1] This is apparent from examination of mean velocity histories for the cardiac cycle, as seen in Figure 10.

A paper by Nerem et al., "An experimental study of the velocity distribution and transition to turbulence in the aorta," (Journal of Fluid Mechanics, Vol. 52, part 1) uses experimental data to derive the critical Reynold's number (for onset of turbulence) in terms of Womersley's frequency parameter $\alpha$. For the descending thoracic aorta,

$$Re_c = 250\alpha$$

since

$$\alpha = a\sqrt{\frac{\omega}{\nu}} \qquad \text{where } a = \text{aortic radius}$$

[1] International Symposium on Pulsatile Blood Flow, D. A. McDonald, pp. 84-96.

increasing α corresponds to decreasing the time available in one cardiac cycle as compared with time required for the amplification of a disturbance. Since the disturbance lacks sufficient time to develop to turbulent proportions, the critical Reynold's number increases (in keeping with Nerem's equation). Similar data for the ascending aorta suggest that

$$Re_c = 150\alpha$$

a lower Reynold's number which is logical in view of the appreciable disturbances already present in this location due to aortic valve closure. It should be noted that Nerem's formulas are not universally applicable, but rather are valid only for blood flow through the aorta.

The complexity of analysis of pulsatile blood flow, and subsequent lack of empirical formulas, is discussed by Streeter (et al).[1] The concept of laminar and turbulent flow is generally applied to steady uniform flow. Where the flow is laminar and the Reynold's number is less than critical, Poiseuille's equation (Appendix A) applies, and the energy dissipation varies as the first power of velocity. Above the critical value, the flow becomes turbulent, and the losses tend to vary as an exponential function of the velocity. As seen in Figures 11 and 12, in the vascular system, except in the proximal aorta, the Reynold's number

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[1] International Symposium on Pulsatile Blood Flow, Streeter et al, pp. 149-177.

is usually well below the critical value of 2000,[1] although some local turbulence is inevitable due to random motion of red blood cells. Streeter also cites the previously-mentioned situation where there is a rapid mixing of dye particles in pulsatile flow even when the Reynolds number at peak velocity is less than critical. Separation of the fluid streamlines near the wall, with each reversal of the pressure gradient, is probably the principal factor which distinguishes pulsatile flow from uniform, steady flow.

Generally speaking, pulsatile flow is characterized by an oscillatory fluctuation superimposed on a mean flow, the oscillatory fluctuation occurring as a function of the systole-diastole piston effect. The effect of the oscillatory fluctuation is dependent upon the vessel's proximity to the heart, since the elastic nature of the large arteries tends to damp out the forward and backward surges created by the heart pump. Moreover, the apparent rise in viscosity with decreasing vessel size (in the arterioles), causes similar damping. D. A. McDonald illustrates two extremes in pulsatile flow in the femoral and saphenous arteries (see Figure 13) and points out the marked reduction in the oscillatory flow components of the smaller saphenous vessel. The fundamental pattern in the major arteries is one with velocity maxima in systole and mid-

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[1] Once again, the arbitrary nature of Re = 2000 is shown by Schultz et al on pg. 91 of Circulation and Respiratory Mass Transport. Transition from laminar to turbulent flow in a bed-ridden, catheterized (cardiac) patient occurred during Systolic ejection at Re = 4400.

diastole and with minima at the end of systole and at the end of diastole. McDonald maintains that although pulsatile flow is not turbulent in the usual sense, it is equally likely that laminar flow is never attained where there is a reversal of the pressure gradient (at the frequency of the arterial pulse) with the separation that results at the wall.

E. O. Attinger[1] adds another dimension in the complexity of blood flow as he points out that the production of turbulence and eddies in the vascular system depends not only on hydraulic depth, Kinematic viscosity and mean velocity, but on frequency and amplitude of the superimposed oscillations, as well as on vascular geometry and the physical properties of the vascular wall. M. G. Taylor examined these variables in a model simulating a set of randomly-bifurcating tubes. He found that the major features of wave transmission in the arterial tree appear to depend on the interacting effects of:

1. non-uniform arterial elasticity, leading to peripheral "amplification."
2. reflected waves leading to maxima and minima at "resonant frequencies."
3. variation in cross-sectional area at branching; in general this is an expansion and leads to a fall in wave amplitude.

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[1] International Symposium on Pulsatile Blood Flow, pp. 179-200.

4. viscous damping arising from the properties of blood and of the arterial wall which leads to attenuation of travelling waves.[1]

Blood flow in the smaller vessels is considerably simplified by the application of lubrication theory, as presented by M. J. Lighthill.[2] Lubrication theory is concerned with fluid motions in which there is a direct balance between pressure gradients and viscous forces, and inertia plays an insignificant role in the balance. The theory is thus applicable to small blood vessels (i.e., $Re < 1$ and $D < 100\mu$). The special type of flow expected when pressure gradients are directly balanced by the viscous forces is one lacking all the characteristics and familiar effects produced by fluid inertia:

1. there is no difference between static and dynamic pressures.
2. there are no centrifugal forces.
3. fluid can negotiate sharp bends without difficulty.
4. no secondary flow is set up due to the curvature of the vessel.
5. motion is generally much less sensitive to vessel geometry.
6. there is no tendency to flow separation.
7. motions at $Re \ll 1$ are completely reversible.

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[1] Hemorheology, "The Influence of the Viscous Properties of Blood and the Arterial Wall Upon the Input Impedance of the Arterial System," M. G. Taylor, pp. 143-147.

[2] AGARD Proceedings #65 July 1970 (See also Circulatory and Respiratory Mass Transport, pp. 85-104).

As stated earlier, the capillary system accounts for roughly 25% of the pressure loss in the circulatory system, considerably less than one might expect. Lighthill attributes this to, and thereby justifies, the lubrication theory. Interactions between red cells, at a given volume concentration (hematocrit), are greatest where they are spinning fastest, that is where fluid vorticity is the greatest (i.e., near the wall). These forces of interaction, probably generated in lubricating layers between spinning deformable cells, give a distribution of internal pressure in the red cell ensemble with a centripital gradient that must help maintain the well documented axial concentration. A consequence of this is the net lowering of resistance in vessels of capillary size, due to the fact that vessel resistance is reduced by lowered viscosity at the wall.

Lighthill has extended this concept to tubes of larger diameter (0.2 cm) and higher Reynold's number for steady flow, oscillatory flow and pulsatile flow. Moreover, lubrication theory is given added credibility in a paper by Mason and Goldsmith,[1] who have shown experimentally that spheres in concentrated suspensions migrate away from the tube wall. This results in a two-phase flow of a central, more viscous core surrounded by a thin peripheral, particle-depleted and less viscous zone, with an attendent decrease in the power dissipated in the tube.

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[1] Circulatory and Respiratory Mass Transport, "The Flow Behavior of Particulate Suspensions," pp. 105-129.

With respect to cardiac flow characteristics (at the other end of the spectrum), D. A. McDonald[1] regards the heart as the reservoir which supplies the tubes emerging from it. In view of the questionable validity of Reynolds number as a gauge of turbulent/laminar flow in a pulsating system, the characteristic of prime interest is the degree of mixing in different areas of the heart. Treating the heart as a wide channel, and considering the contractile nature of the heart, together with the irregular shape of the channel near the valves, there is little doubt that the resultant churning flow throughout causes thorough mixing in all four chambers. This fact has been verified by means of intracardiac catheters, and it is generally agreed that the blood is thoroughly mixed by the time it reaches the pulmonary artery. The significance of this churning action vis-à-vis hemolysis considerations for an artificial heart, will be discussed later.

As pointed out earlier, with vessel size as the criterion turbulent flow is generally restricted to only the largest arteries while laminar flow predominates the remainder of the circulatory system (see Fig. 11). The difficulty encountered in this categorization lies in the uncertainty as to whether "turbulent" flow is really turbulent, or merely due to eddies and unsteady effects of pulsatile flow and motion of red blood cells. While such

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[1] Blood Flow in Arteries, pp. 58-59.

gages as Reynolds number, arterial catheterization and murmurs do not dispel this uncertainty, they do serve as an indication of the effects of curvature, branching and valves on blood flow characteristics.

McDonald notes that the effect of curving a pipe is to increase the stability of flow, the critical Reynold's number increasing quite markedly to a value of 7000. Studies of steady flow through a full-scale model of the arch of the human aorta were made by Timm in 1942.[1] At rates of flow that caused turbulence in the descending aorta, Timm's photographs showed the flow in the proximal part of the arch to be clearly laminar, although the streams of dye pursued a somewhat complicated helical course. These helices are due to the centrifugal force associated with motion in a curve, this force being greatest in the fast-moving axial streams, and least in the slow-moving streams near the wall. Two minor (secondary) circulations are set up as the fluid in the middle of the stream forms a secondary flow toward the outer wall, forcing the fluid near this wall to move towards the inner wall of curvature (see Fig. 14).

Further influence of vessel geometry on flow characteristics is discussed by D. A. McDonald,[2] with regard to junctions and valves. The junctions are divided into three main types:

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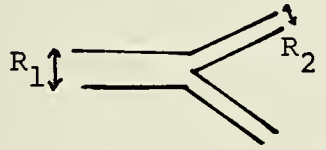
[1] Ibid., pp. 60-61.

[2] Ibid., pp. 61-67.

1. Bifurcation (Y-junction) where the main trunk is divided into two (or more) equal branches;
2. Side branch, where a single branch, usually of smaller dimensions, leaves the parent trunk at an angle;
3. Fusion (Λ junction), where two or more branches of equal size join to form a trunk.

In the first two cases the blood is flowing from a region of higher Reynold's number to one of lower Reynold's number, since




$$\frac{Re_1}{Re_2} = \sqrt{nd}$$

where n = number of branches

$$d = \frac{nA_2}{A_1}$$

and the total branch cross-sectional area is greater than that of the parent trunk. Thus, the effect is to increase the stability of flow, dependent on the branching angle. The branch angle must be considered since there is a discontinuity in the wall at the juncture, creating eddies (see Fig. 15). If these eddies are strong enough, they may persist long enough to create a lingering disturbance to offset the increased stability due to this branching. In the third case, the fusion junction, flow is from an area of lower to higher Reynold's number. At rates of flow

considerably below the critical Reynold's number (600 in branches, 1000 in main trunk) it was observed in a glass flow model that vortex rings were formed at this junctional region and were carried down the pipe, showing no signs of damping out for the length of some 30 diameters that they were followed. This strongly suggests a destabilizing effect of the fusion junction.

The principal effect of valves is that they cause eddies to form; if the disturbance is small the eddies will soon die away, but if large, the eddies will grow in size and ultimately cause a complete breakdown of laminar flow. McDonald notes that the valve cusp is the commonest natural projection, and develops a crude approximation for cusp size as a determinant in the transition from laminar to turbulent flow in valve passage. In doing so, he utilizes Goldstein's formula for sharp-edged projections, and treats the valve cusp as a single roughness element. Goldstein's formula for a sharp-edged projection of height $\varepsilon$ in a pipe of radius r then becomes:

$$\frac{\varepsilon}{r} < \frac{4}{(Re)^{\frac{1}{2}}}$$

is necessary to maintain laminar flow. At Re of 400, for example, the tolerated ratio of $\varepsilon/r$ is 1/5 and at Re = 1600, $\varepsilon/r$ for laminar flow is 1/10.[1]

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[1] Blood Flow in Arteries, D. A. McDonald, pp. 65-66.

## VI. PHYSIOLOGICAL DISORDER OF FLUID DYNAMIC ORIGIN

By and large the most serious disorders in the circulatory system are those located in the heart. Among these are congenital septal defects, valves damaged by rheumatic fever, and coronary insufficiency. In contrast to the foregoing disorders, which are relatively localized, man is also plagued by disorders of systemic proportion.

Arteriosclerosis, or hardening of the arteries, is a degenerative disease whose origin is fluid dynamic in nature.[1] It appears to be associated with the deposition of calcium in the arterial walls, the net effect being a resultant decrease in the flexibility of the affected vessels. When the arteries lose their flexibility, the Windkessel effect is lost, so the heart must pump harder and at higher pressures. These higher pressures may cause problems in the brain where a ruptured vessel will cause a cerebral hemorrhage or stroke. By the same token, when the blood pressure is consistently too high (hypertension) damage to the heart, blood vessels or kidneys may result.

Another problem arises out of the body's defense mechanism, clot formation, when the vessel lining is injured by wound, infection or chronic disease. The formation of clots occurs in four major steps. Immediately, after a blood

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[1] Biology, Goodnight et al., pp 68-69.

vessel is cut or ruptured, the wall of the vessel contracts, thereby instantaneously reducing the outflow of blood. Next, the platelets attempt to plug the rent in the vessel, adhering to the endothelial wall which has lost its natural non-wettability at the site of the trauma. On adhering, the platelets themselves take on new characteristics, the most important being that they become sticky to other platelets. In this manner, layers of platelets adhere to one another until the rent is finally filled. The third step is the formation of the clot itself, a vessel-occluding process as opposed to the rent-plugging process of the platelets. This step is characterized by the formation of a fibrin clot which, when invaded by fibroblasts, becomes the basis of the fourth step--namely the growth of fibrous tissue into the blood clot to close the hole in the vessel permanently.[1] These thrombi (clots) may grow to occlude the entire vessel and may even break loose (then called emboli) and become lodged in some smaller vessel. The effects of the thrombus will depend on the vessel obstructed: a sudden paralytic stroke when the blood supply to part of the brain is stopped; an acute heart attack, perhaps death, when a branch of the coronary artery is plugged (coronary thrombosis) and heart muscle is deprived of blood; death of tissue masses and gangrene when the circulation in a limb or an internal organ is

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[1]Guyton, Textbook of Human Physiology, pp. 136 and 137.

blocked.[1] This process of thromboembolism is depicted in Figure 16.

In a paper delivered before the Advisory Group for Aerospace Research and Development, Caro, et al[2] discusses the role of shear rate in the arterial distribution of early atheroma. It has been found that early atheroma develops preferentially in those regions which experience relatively low wall shear, inconsistent with a mechanical wall damage hypothesis. The authors therefore propose that wall shear has a controlling (inhibiting or retarding) rather than a causative influence on atherogenesis. In developing the mathematical gauge of wall shear, the authors assume the arterial flow is steady, that the arterial walls are rigid, and that blood is a Newtonian fluid. These assumptions are justified inasmuch as the slight pulsatile expansion and relaxation of the truly flexible arteries produces almost insignificant radial velocities, hence little effect on wall shear. Moreover, although blood is actually a non-homogeneous fluid, the shear rates obtained in the larger arteries make the Newtonian approximation acceptable. Finally, due to typical Reynold's numbers and arterial lengths, it is quite likely that the flow is entrance length in type, i.e., thin boundary layers and relatively high wall shear rates. The latter situation

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[1] The Body Functions, Gerard, pp. 131-132.

[2] AGARD Conference Proceedings No. 65, pp. 13-1 thru 13-8.

fosters a tendency for boundary layers to thicken and wall shear rates to increase with distance from the branch.

In keeping with Caro's hypothesis, experimental study of flow at junctions shows the flow divider to be a surface on which new boundary layers must grow (hence a region of higher shear) while the outer wall of the junction experiences relatively low shear. Accordingly, the greatest concentration of early lesions is coincident with regions experiencing relatively low shear. Studies of the visceral branches of the abdominal aorta reveal a relative sparing of the flow dividers and relatively heavy involvement of the outer walls at these junctions. This is demonstrated in Figure 17 where one outer wall is in a "dead-water" region, i.e., one of low shear.

## VII. PROSTHETIC DEVICES

All of the bioengineer's efforts to understand the fluid dynamics of the circulatory system are rewarded when he is called upon to design a replacement for some portion of this system. In a highly informative paper[1] Kuchar and Scala indicate the scope of this design problem, consisting of problems in the areas of materials, control systems, power supply and transmission, and hemodynamics, all of which are currently under investigation in many laboratories. Problems in blood flow arise because of the complex nature of the interaction between flowing blood and the material surfaces which are introduced into the flow system. These include destruction of the red blood cells (hemolysis) and other formed elements of the blood, thrombus formation, clotting, and rejection.

In regard to the design of prosthetic devices, clinical studies of the cardiac function have been carried out in millions of patients[2] so that many operating characteristics of these organs are now well known and documented. Kuchar[3] defines an optimum design as one in which the cardiac assist device performs the systemic function without

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[1] Design of Devices for Optimum Blood Flow, Kuchar & Scala.

[2] See, for example, An Outline of the Clinical Physiology of the Circulation, Parin & Myerson NASA TT F 173.

[3] Design of Devices for Optimum Blood Flow, Kuchar & Scala.

significant injury to the host at the lowest cost. This definition implies a series of trade-offs or engineering design compromises in the consideration of the following questions:

1. What are the desirable size and weight of the device?
2. Will it be implanted or used as an extracorporeal device?
3. What is the nominal design point of the device in terms of flow rate, pressure level, operating temperature, cycle frequency, waveform, etc.?
4. What are the permissible upper and lower levels for the aforementioned parameters?
5. What types of power source and transmission methods are needed and are available?
6. What types of controls are needed?
7. What type of cooling is required?
8. What materials are compatible with body tissue and blood?
9. What is the desired operating life of the device?
10. What is the maximum allowable cost in order to permit the widest possible usage?

The replacement of a failing human heart is a surgical technique the mechanics of which have already been perfected. The major difficulties lie in post-operative complications such as immunization rejection (immune reaction) of the transplanted heart, or the overall physical weakness of the

recipient under the influence of massive doses of drugs to suppress this reaction. These facts, together with the shortage of "donor" hearts, dictate the development of a heart assist device or completely mechanical heart. For this reason, we will concentrate on the hemodynamic problems encountered in the design of

1. Heart lung machines
2. Cardiac assist devices
3. Cardiac replacement devices

Runge, et al[1] define the permanent implanted assist device as one in which the patient's heart is left intact, the device being designed to assume a portion of the workload of the failing ventricle or ventricles. In contrast, the total heart replacement device is substituted for the patient's heart when the latter is damaged to the extent that partial cardiac assist is no longer feasible.

Of the three categories, the heart lung machine presents the fewest problems. Because of the temporary and extracorporeal use of the device, there are no constraints on its size and weight, power supply or control system; furthermore, the problems of hemolysis and thrombus formation are somewhat less critical than for permanent or implantable devices because of the availability of transfusion blood and the relatively short periods of operation. Current heart-lung machines employ rotating disc, rotating screen,

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[1] "Permanent Implanted Cardiac Assist Device and Total Cardiac Replacement Device," AD 690135.

or bubble oxygenators and a roller or finger-type pump which compresses the elastic walls of the blood-carrying tube, thus providing a forward flow. The peristaltic pumping mechanism of the roller pump is one where the roller (or mechanical fingers) occlude the tube and "milk" the fluid through it. This type pump has the advantage of segregating the transported fluid from the mechanical parts of the pump. The disadvantage lies in the fact that although complete occlusion is not necessary for the pump to be effective, very high stresses are concentrated around the point of minimum radial gap in the tube. These stresses, in turn, hemolyze blood at a much higher rate than the normal in vivo rate. With this in mind, Jaffrin and Meginniss[1] performed a mathematical analysis of occlusive vs. non-occlusive pumps vis-à-vis hemolysis rate. While lacking any specific design recommendations, their study lends mathematical support to the intuitive expectation that a design which reduces the high shear stresses as well as the fraction of blood flow submitted to them will also reduce hemolysis.

The heart-lung machine is not suitable for prolonged operation because the problems of hemolysis and clotting become increasingly critical. For example, due to surface chemistry effects at the blood-gas interface in the oxy-generator, severe damage to the protein constituents of the

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[1] The Hydrodynamics of Roller Pumps and their Implication in Hemolysis, pp. 1-18.

blood can occur. Moreover, large shearing stresses and turbulence caused by the pump also result in hemolysis (the best available pumps causing this cellular destruction at a rate about 1/3 greater than the human heart).[1] Hemolysis, in turn, can lead to progressive anemia or the inability of the body to tolerate the free hemoglobin introduced from the damaged red cells into the blood plasma. Finally, clotting of the blood on the surface of the oxygenation and pumping equipment can become a long-term problem.[2]

While the chemical process of thrombus formation on an artificial surface is somewhat different from that on the blood vessel wall, the mechanics are quite similar. The triggering mechanism is presumed to be some property of the surface imperfection (or inhomogeneity) at which the thrombus formation occurs.. The first microscopically observable phenomenon in the sequence is the adhesion of individual platelets to the surface (step 2 in in vivo clotting). The small delay for this to occur is attributed to the time required for a layer of protein to be adsorbed onto the artificial surface. This protein layer serves as the anchor for subsequent platelet aggregation, since the protein layer is so modified as to become attractive to platelets.

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[1] See "Red Blood Cell Damage by Shear Stress," (in Biophysical Journal Vol 12, 1972) for a discussion of this problem. Leverett et al examine experimentally the degree of hemolysis caused by the following factors: 1) interaction of erythrocytes with solid surfaces, 2) centrifugal force, 3) damage at air-blood interfaces, 4) cell-cell interaction, 5) viscous heating.

[2] Design of Devices for Optimum Blood Flow, Kuchar & Scala. pg.5.

Moreover, the protein layer (separating the surface from the blood) may be the factor which explains experimental evidence that the platelet deposition rate is not a strong function of the type of surface. Once past the deposition of a monolayer of platelets, platelet aggregation commences and the thrombus formation process is well established.[1] As noted earlier, if the thrombi break away from the surface without being dissolved (by the anti-coagulent known as plasmin), there exists the probability of some blood vessel occlusion, which in the extreme could cause a stroke or even death.

The development of complete artificial hearts, totally replacing the heart function on a long time basis, is a monumental task as described by Kuchar and Scala.[2] In addition to the requirements on pressures and flow rate, the device must pump from 1 to 2 gallons per minute, and must have two separate ventricles or pumping chambers; the first, pumping blood to the lungs, must develop pressures of from 20 to 80 mm Hg, while the second, pumping blood through the remainder of the body, must develop pressures of about 120 to 180 mm Hg. In addition, the flow rates of both pumping chambers must be equal. These devices should

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[1] The Fluid Mechanics of Thrombus Formation, pp. 3 and 4.

[2] Design of Devices for Optimum Blood Flow, Kuchar and Scala, p. 6.

be implantable in the chest cavity, and hence must satisfy stringent requirements as to size, weight, reliability (normally a 10 year lifetime with virtually no maintenance possible), compatability with body tissues, heat output, and hemolysis rate.[1]

Given a reasonable heat output by the device, the human body is well-equipped to handle the dissipation of this additional heat. The skin, the subcutaneous tissues, and the fat of the subcutaneous tissue represent a heat insulator for the body and are designed to maintain a normal internal temperature regardless of the climate. A. C. Guyton refers to the flow of blood to the skin as the "radiator" system of the body. Heat conduction to the skin by the blood is controlled by the degree of vasoconstriction of the arterioles that supply blood to the venous plexus underlying the skin. The degree of vasoconstriction, in turn, is determined by the sympathetic nervous system (specifically, the hypothalmus) which is tonically active, causing continual constriction of the arterioles in the venous plexus. It is presumed that there are temperature receptors in some internal organs which transmit signals ultimately to the posterior centers of the hypothalmus when overheated, thereby decreasing its activity and the degree

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[1]Runge et al., in Perm. Implanted Cardiac Assist Device and Total Cardiac Replacement Device, emphasize the necessity for completely enclosing the device with no external connections, to preclude any introduction of infection along the route of the exterior wire or tubing.

of vasoconstriction. The rate of blood flow under conditions of vasodilatation can be as high as 30% of the total cardiac output, and the associated high rate of blood flow causes heat to be conducted from the internal portions of the body to the skin with great efficiency. The only expense in this process is borne by the heart by way of increased cardiac output, but this would presumably be included in the design operating limits of the mechanical heart.

An indication of the amount of experimental research underway in this area is given by editors Nose and Levine[1] in a collection of abstracts on the state of the art in artificial heart development. The abstracts cover such wide-ranging possibilities as:

1. an electromagnetic heart
2. a pneumatic (air-driven) heart
3. a heart powered by an electrolytic power plant
4. a heart driven by a nuclear-powered steam engine

Some of the major problems encountered, similar to those outlined by Kuchar and Scala are:

1. clotting of blood on the prosthetic heart surfaces which became a fatal development in most all the experiments
2. dissipation of the power plant heat
3. size and weight limitations

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[1] Advances in Biomedical Engineering and Medical Physics, Vol. 3, "Cardiac Engineering."

While the problem of hemolysis is one primarily of design, there is considerable activity underway in the area of blood-material interaction in an attempt to eliminate the clotting problem. Newer polymers which are non-reactive with blood (e.g., the celanese product Celcon), together with the process of heparinization (a medicated coating to prevent rouleaux formation) may reduce hemolysis when applied to artificial flow surfaces.

If one considers the cardiac assist device, then the power plant and all its associated problems are eliminated. This alternative is feasible only if the heart ventricles are merely weak or failing, and can function normally with some artificial assist. The most common adaptation is a left atrial to aortic bypass, to remove part of the volume load from the left ventricle, and the other adaptation of such a device is a right atrial to pulmonary artery bypass to assist the right ventricle in its work-load. The device itself utilizes the respiratory motion of the rib cage as the driving mechanism. As outlined by Runge et al[1] the device is a compression pump, being fixed to the posterior left thoracic cage in the sulcus behind the heart with connections to the left atrium above and the aorta below (see Figs. 18, 19 and 20). The aorta above the device is banded to approximately 1/3 its usual diameter, to prevent overloading the left ventricle during the systolic

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[1] Permanent Implanted Cardiac Assist Device, Runge et al, pp. 11-16.

phase of the assist device. This potential overload exists due to the disparity between the respiration (the assist device driving force) at 16/minute and the normal resting heart rate of 60 or more per minute. In effect, then, the device supplies the major arterialized flow to the body below the diaphragm, while the patient's left ventricle supplies the major output to the head and upper extremities. The stroke volume of this device, operating off the thoracic cage motion, is estimated at 200 cc, so that at a respiratory rate of mild distress of 30 per minute, its output would be 6000 cc/min. The device thus offers the advantage of increasing its output with increased respiratory effort and rate.

## VIII. CONCLUSIONS AND RECOMMENDATIONS

Some conclusions may be drawn on the two bodies of information considered in this paper, namely analytic models and empirical measurements. Dealing with the first, an exhaustive literature survey has shown that there is no one flow model which can simulate blood flow in the various parts of the human body. Womersley's equations apply well to large arteries and veins where flow is generally considered pulsatile and periodically turbulent, and the blood is generally accepted to be Newtonian in nature. At the other end of the flow scale, the microcirculation, such phenomena as bolus flow and the lubrication theory enable one to deal with capillary flow as being steady and laminar, with blood considered Newtonian in nature. Here, then, is where Poiseuille's Equation applies. The middle ground (i.e., smaller venules and arterioles) is the more complex flow regime, the flow being pulsatile, possibly turbulent (dependent on vessel geometry) and the blood non-Newtonian in nature. The Casson fluid model best describes the latter flow regime. The foregoing simplification of human blood flow into three models belies the "grey" areas linking the three, as determined by variations in such parameters as hematocrit, shear rate and vessel geometry and structure. Nevertheless, the three models provide the engineer with a remarkably good mathematical description of blood flow in the major part of the human circulatory system.

The research aspect in cardiovascular engineering is directly related to the aforementioned flow models. For example, the flow model will predict the shear rate present at a particular location in a blood vessel, and this information is vital in determining to what extent thromboembolism is a design consideration for a prosthetic device in this location. In the research field, some of the most serious problems encountered in the use of prosthetic devices are clotting, hemolysis and rejection. The permanently implanted cardiac assist device is one of the best alternatives, in that the turbulence and shear rate associated with its location are such that clotting is not a problem. Hemolysis is reduced since the pumping load of the device, as an assistant, is of smaller magnitude than that of a replacement device. Finally, the fact that it is a bypass to the normal circulatory route minimizes the interference aspect of the assist device, thereby eliminating many problems.

There are many potential routes for further thesis research in this area. For example, engineers have recently designed an atomic-powered cardiac replacement device. While not much information has yet been published as to the success of this device, it is certain that the problems of clotting, hemolysis and rejection are still current considerations. Moreover, the problem of power plant heat dissipation will most probably be of greater significance than that of alternative power plants. For example, if the

human body is left to cope with this heat, what effect will the continued vasodilatation have on the circulatory system?

Other areas of continuing interest are the development of immuno-suppressive drugs to eliminate the ironic problem of the body's rejection of a device designed to prolong its existence. While this subject has definite biological overtones, a student interested in chemistry might examine the effectiveness (and longevity) of chemical coatings on prosthetic surfaces to eliminate the often-fatal complication of thromboembolism. Finally, considerable research is being conducted to develop materials more compatible with the human body, and less reactive with blood, a dual-purpose project to eliminate the problems of rejection and hemolysis. Continuing interest and work in this field is sure to overcome the obstacles which stand between man and his first step toward immortality.

# APPENDIX A

# FLOW MODELS

## A. POISEUILLE FLOW

In 1842, Poiseuille published the first experimental work indicating that the volume flow rate in cylindrical tubes was proportional to the fourth power of the diameter:[1]

$$Q = \frac{KPD^4}{L}$$

where Q = flow volume
P = pressure drop along the tube
L = length of the tube
D = diameter of the tube

The constant K was observed to fall with increasing temperature for liquids, and was determined to be a measure of viscosity.

This formula was generally restricted to Newtonian fluids in steady, laminar flow and could not, therefore, be applied to blood flow due to the anomalous viscous properties of blood in unsteady motion. Weidemann (1856) and Hagerbach (1860) later showed the constant to be:

$$K = \frac{\pi}{128\mu}$$

where $\mu$ is the dynamic viscosity so that

---

[1]Note that these results can be obtained from the exact solution of the Navier-Stokes equations.

$$Q = \frac{(P_1 - P_2)\pi a^4}{8\mu L}$$

where a = tube radius. Note also that the tube diameter does not change with pressure, ignoring the elastic properties of the vessel wall.[1] Figure 21 compares velocity vectors and streamlines for flow in a rigid-wall vessel with those for flow in a viscoelastic vessel.

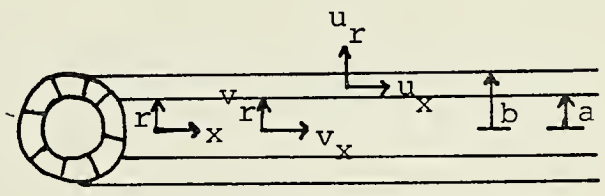
## B. WITZIG (1914) AND WOMERSLEY (1955)

The most realistic model for pulsatile blood flow is that representing wave propagation in a distensible tube containing viscous fluid. The first analysis of this kind was done by Witzig in 1914, starting with linearized Navier-Stokes equations:

$$\left( \frac{V_x}{C}, \frac{V_r}{C} \ll 1, \; C = \begin{matrix} \text{wave} \\ \text{speed} \end{matrix} \right)$$

Continuity

$$\frac{\partial V_x}{\partial x} + \frac{\partial V_r}{\partial r} + \frac{V_r}{r} = 0$$




Momentum (axial)

$$\frac{\partial V_x}{\partial t} = -\frac{1}{\rho}\frac{\partial P}{\partial x} + \nu \left\{ \frac{\partial^2 V_x}{\partial r^2} + \frac{1}{r}\frac{\partial V_x}{\partial r} + \frac{\partial^2 V_x}{\partial x^2} \right\}$$

Momentum (radial)

$$\frac{\partial V_r}{\partial t} = -\frac{1}{\rho}\frac{\partial P}{\partial r} + \nu \left\{ \frac{\partial^2 V_r}{\partial r^2} + \frac{1}{r}\frac{\partial V_r}{\partial r} + \frac{\partial^2 V_r}{\partial x^2} - \frac{V_r}{r^2} \right\}$$

---

[1] McDonald, D. A., Blood Flow in Arteries, pp. 13-15. See also Rheology of the Circulation, Whitmore, pp. 39-42.

where $\rho$ = fluid density
$\nu$ = fluid kinematic viscosity
$P$ = the pressure
$V_r$ = radial velocity component
$V_x$ = axial velocity component
$r$ = radial coordinate
$x$ = axial coordinate
$t$ = the time

These equations were developed based on the assumption that fluid motion was laminar and axially symmetric, and that the fluid itself was incompressible, homogeneous and Newtonian.

Thin-walled tubes required the application of membrane theory equations, but for simplicity's sake, we will concern ourselves only with thick-walled tube (approximating the larger arteries). The thick-walled tube is assumed to be a linear viscoelastic, isotropic and incompressible material and the equations of motion for this material are:[1]

$$\frac{\rho_w}{\mu^*}\frac{\partial^2 u_r}{\partial t^2} = \frac{\partial^2 u_r}{\partial r^2} + \frac{1}{r}\frac{\partial u_r}{\partial r} - \frac{u_r}{r^2} + \frac{\partial^2 u_r}{\partial x^2} - \frac{1}{\mu^*}\frac{\partial \Omega}{\partial r}$$

$$\frac{\rho_w}{\mu^*}\frac{\partial^2 u_x}{\partial t^2} = \frac{\partial^2 u_x}{\partial r^2} + \frac{1}{r}\frac{\partial u_x}{\partial r} + \frac{\partial^2 u_x}{\partial x^2} - \frac{1}{\mu^*}\frac{\partial \Omega}{\partial r}$$

$$\frac{\partial u_x}{\partial x} + \frac{\partial u_r}{\partial r} + \frac{u_r}{r} = 0$$

[1] "Wave Propagation Through a Newtonian Fluid Contained within a Thick-walled viscoelastic tube," R. H. Cox, Biophysical Journal, 6 March 1968.

where $\rho_w$ = density of wall material
$\mu^*$ = wall modulus of rigidity
$\Omega$ = a finite pressure
$u_x$ = axial component of wall displacement
$u_r$ = radial component of wall displacement

The boundary conditions, coupling the fluid and tube motions, involve continuity of stress and velocity components at the fluid-tube interface and are

1. The fluid velocity components are finite at r = 0, i.e.

$$V_r = 0$$

$$\frac{\partial V_x}{\partial r} = 0$$

2. The axial and radial velocity components of the fluid and wall are continuous at the boundary r = a, i.e.

$$V_r = \frac{\partial u_r}{\partial t}$$

$$V_x = \frac{\partial u_x}{\partial t}$$

3. The stress components of the fluid and wall are continuous at the boundary r = a, i.e.

$$\mu\left\{\frac{\partial V_r}{\partial x} + \frac{\partial V_x}{\partial r}\right\} = \mu^*\left\{\frac{\partial u_r}{\partial x} + \frac{\partial u_x}{\partial r}\right\}$$

$$-P + 2\mu \frac{\partial V_r}{\partial r} = -\Omega + 2\mu^* \frac{\partial u_r}{\partial r}$$

4. The stress components in the wall at r = b are zero, i.e.

$$\mu^* \left[ \frac{\partial u_r}{\partial x} + \frac{\partial u_x}{\partial r} \right] = 0$$

$$-\Omega + 2\mu^* \frac{\partial u_r}{\partial r} = 0$$

Witzig thus laid the groundwork for the mathematical model equations, the solution of which eliminated the Poiseuille restriction to steady flow conditions. In 1955, Womersley utilized these governing equations and boundary conditions to develop a solution for volume flow rate in a form that was easily computed:

$$Q = \frac{\pi a^2 A}{\mu i^3 \alpha^2} \left\{ 1 - \frac{2J_1(\alpha i^{3/2})}{\alpha i^{3/2} J_o(\alpha i^{3/2})} \right\} e^{i\omega t}$$

where $J_o$ and $J_1$ are Bessel functions and

$$\alpha = a\sqrt{\frac{\omega}{\nu}}$$

is the Wormsley frequency parameter

a = tube radius

A = Fourier coefficient for simple harmonic motion

$\omega$ = circular frequency simple harmonic motion

Womersley further computed tables of values for the {braced} expression, the parameter $\alpha$ and Fourier coefficient A, based on the harmonic at which oscillation occurs (see pages 293 and 296, Appendix I in McDonald's Blood Flow in Arteries).[1]

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[1] Blood Flow in Arteries, D. A. McDonald, pp. 81-82.

C. CASSON FLUID

Wormersley's work on pulsatile flow was based on a Newtonian fluid, and recent work has shown that this simplified flow model is not a suitable representation of blood flow in smaller vessels.[1] A more physiologically relevant model for the blood in this flow regime is the Casson fluid. The Casson fluid is one having a finite yield stress and shear-dependent viscosity, and whose constitutive relations are given by:

$$(1) \qquad \tau^{\frac{1}{2}} = \tau_y^{\frac{1}{2}} + \mu^{\frac{1}{2}} \left(\frac{\partial \tilde{u}}{\partial \tilde{r}}\right)^{\frac{1}{2}} \quad \text{if} \quad \tilde{\tau} \geq \tau_y \qquad \text{(Newtonian)}$$

and

$$(2) \qquad \frac{\partial \tilde{u}}{\partial \tilde{r}} = 0 \qquad \text{if} \quad \tilde{\tau} \leq \tau_y$$

where $\tilde{\tau}$ = shear stress

$\tilde{u}$ = axial velocity

$\mu$ = dynamic viscosity

$\tilde{r}$ = radial distance

$\tau_y$ = yield stress[2]

Equation (2) corresponds to the vanishing of velocity gradients in regions where the shear stress $\tilde{\tau}$ is less than

---

[1] However, as stated on page ( 34 ) of the text, if there is an appreciable cell-free wall layer, the Newtonian properties of this lubricating wall layer may outstrip the cell-rich non-Newtonian core (plug) in its effect on gross flow characteristics.

[2] Whitmore, in Rheology of the Circulation, p. 84, defines yield stress as that stress required to break the connections linking (blood) constituent particles before continuous flow can commence. The implication here is that yield stress is closely linked to hematocrit.

the yield stress $\tau_y$ (i.e., plug flow, as described on page 13 of the text). When the shear rates in the fluid are very high, i.e., $\tilde{\tau} > \tau_y$ as in equation (1), then the Newtonian behavior is indicated, as suggested on page 9 of the text. Such behavior is attributed to the flow of blood in small tubes.[1]

Consider the flow of the Casson fluid in a sufficiently long rigid tube under the influence of a periodic pressure gradient. It is assumed that entrance, end and special wall effects are not present, that the fluid is incompressible and the pressure gradient depends only on time. For such flows, the axial component u is the only component of velocity, and is zero at the tube wall. The simplified momentum equation, in non-dimensional form, is

$$(3) \qquad \alpha^2 \frac{\partial u}{\partial t} = - P(t) + \frac{1}{r} \frac{\partial}{\partial r} (r\tau)$$

where

$$\alpha^2 = \frac{a^2 \omega}{\nu}$$

Since $\tau$ is a function of velocity gradient, equations (1) and (2) in non-dimensional form are

$$(1a) \qquad \tau^{\frac{1}{2}} = \theta^{\frac{1}{2}} + \left(\frac{\partial u}{\partial r}\right)^{\frac{1}{2}} \qquad \text{if} \quad \tau \geq \theta$$

$$(2a) \qquad \frac{\partial u}{\partial r} = 0 \qquad \text{if} \quad \tau \leq \theta$$

where $$\theta = \frac{\tau_y}{(P_o a)/2}$$

---

[1] Aroesty and Gross, The Mathematics of Pulsatile Flow in Small Vessels, p. 3.

and $a$ = tube radius

$P_o$ = absolute magnitude of a typical pressure gradient

For the range of vessels where the Casson Fluid might be a suitable flow model (i.e., low shear, non-Newtonian) the Womersley frequency parameter will be very small. At typical values of vessel radius, viscosity and frequency,

$$\alpha^2 = \frac{a^2\omega}{\nu}$$

is of the order ($\alpha^2 = .01$), hence a solution to equation (3) may be obtained for $\alpha^2 \doteq 0$ as a reasonable approximation to the flow situation.[1]

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[1] Pulsatile Flow in Small Blood Vessels, Aroesty and Gross, pp. 7-9.

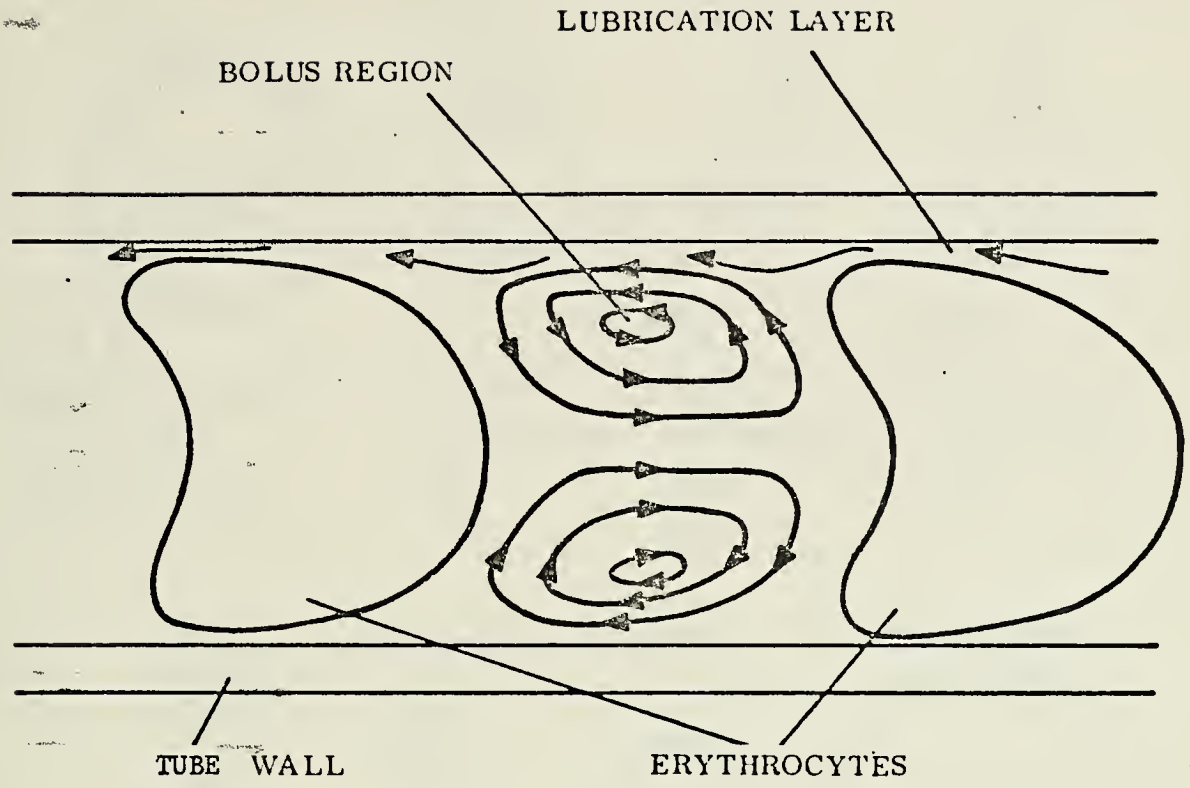



Figure 1. Blood Flow Regions in Erythrocyte-sized Tubes.

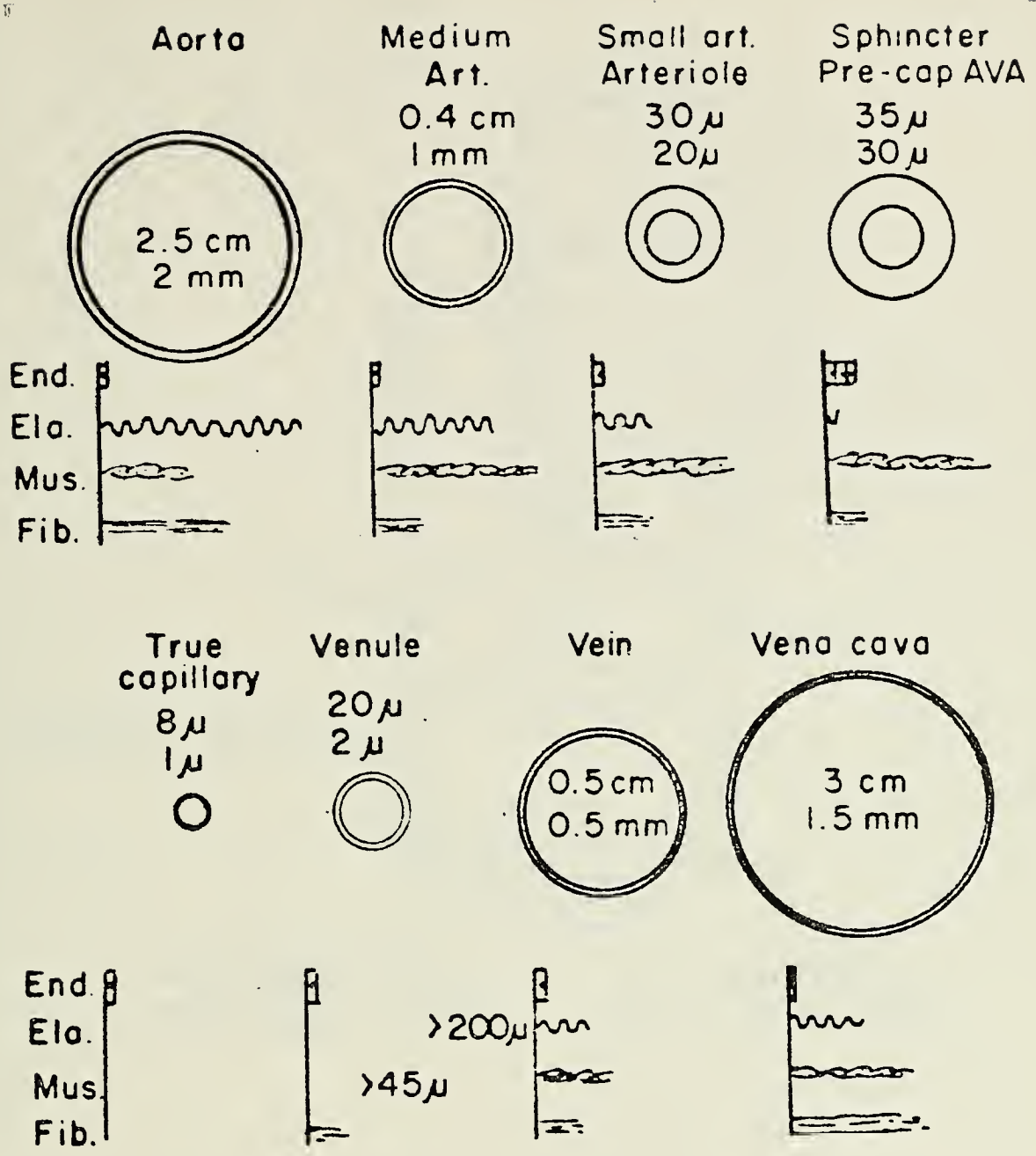



Figure 2. Size, Thickness of Wall and Four Basic Tissues in the Wall of Different Blood Vessels. The figures directly under the name of the vessel represent the diameter of the lumen; next, the thickness of the wall. End. = endothelial lining cells. Ela. = elastin fibers. Mus. = smooth muscle. Fib. = collagenous fibres. (From Burton, A.C. 1944. Relation of structure to function of tissues of the wall of blood vessels. Physiol. Rev. 34: 619-42).

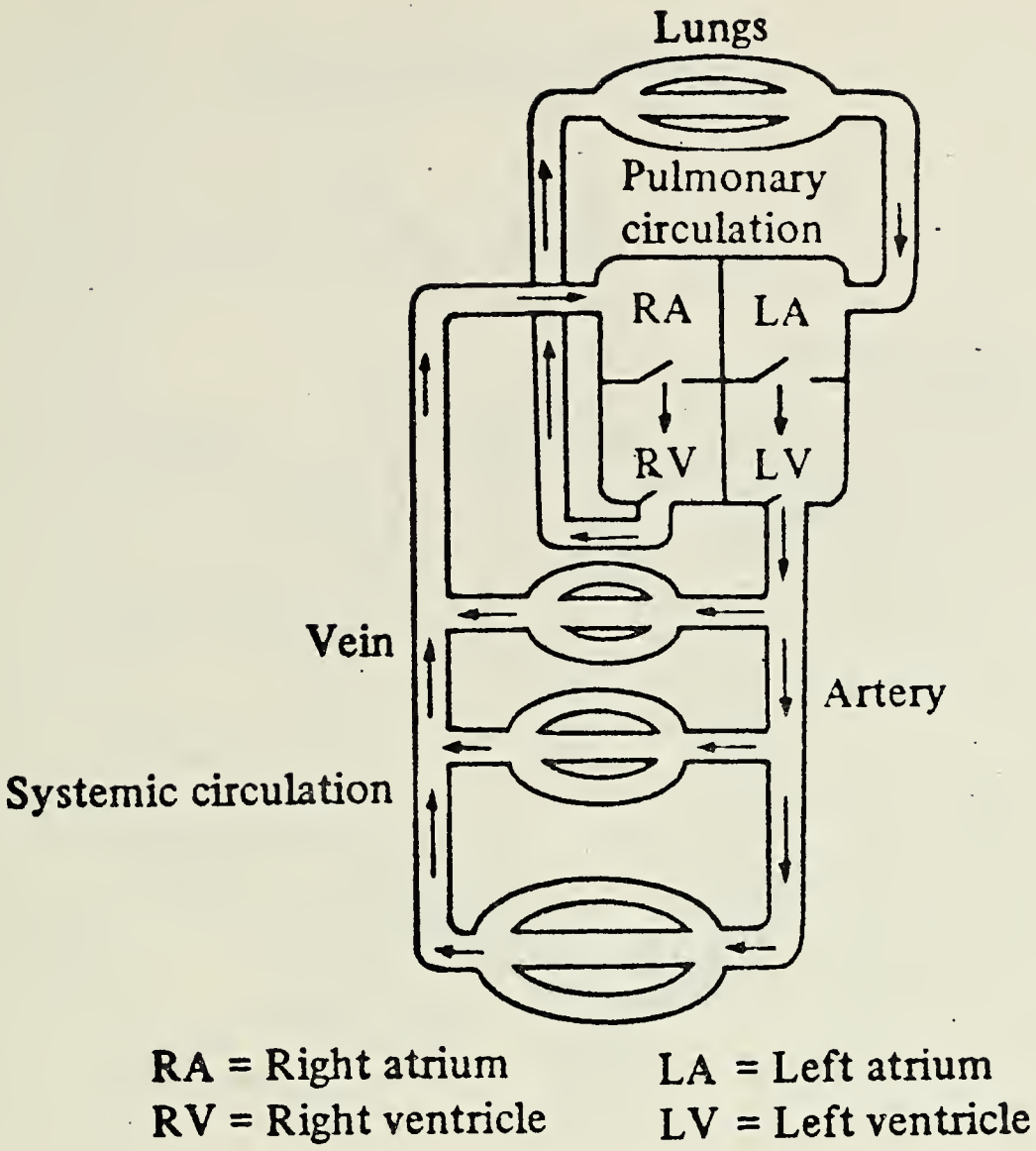



Figure 3. Simplified Schematic of the Circulatory System.

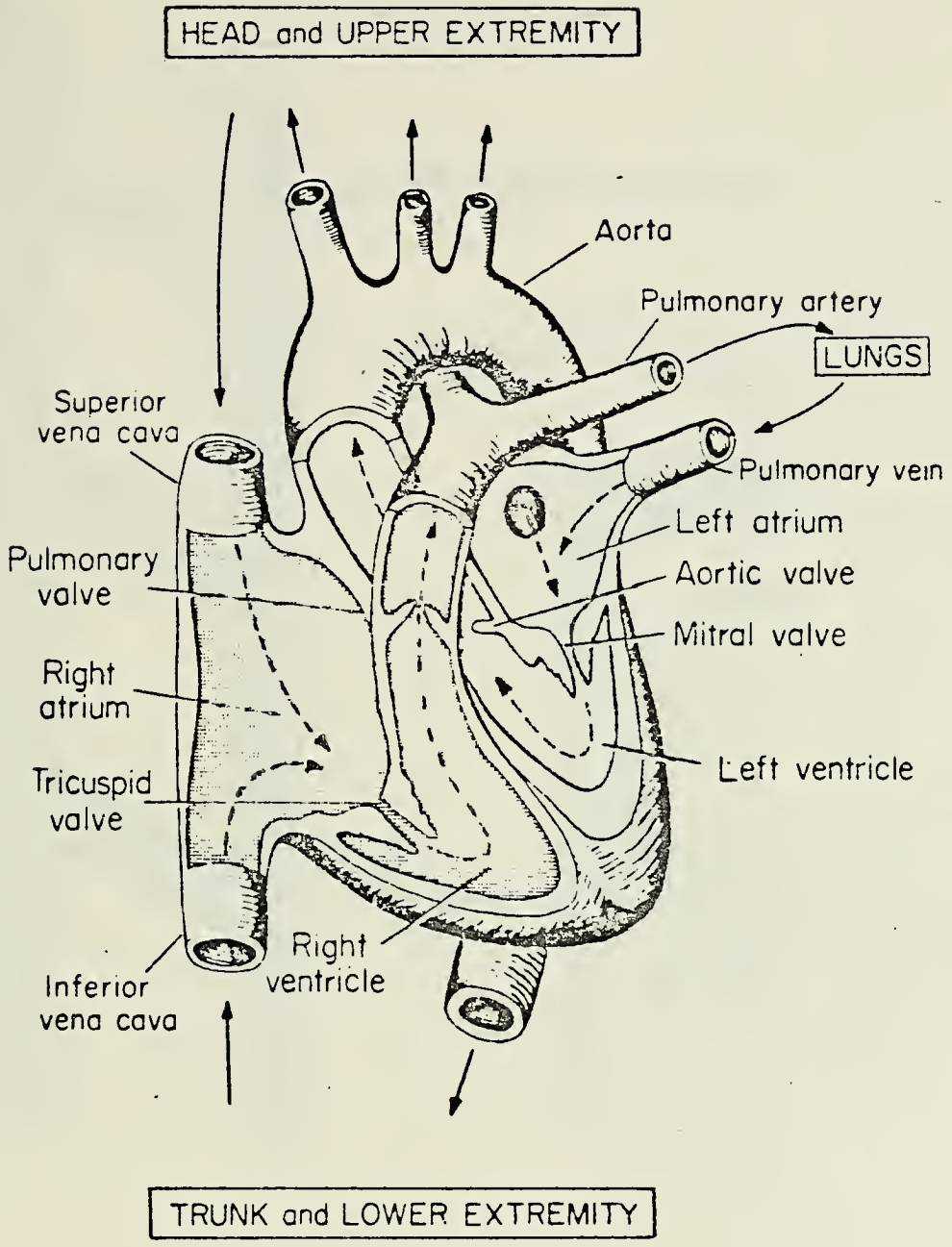



Figure 4. The Functional Parts of the Heart.

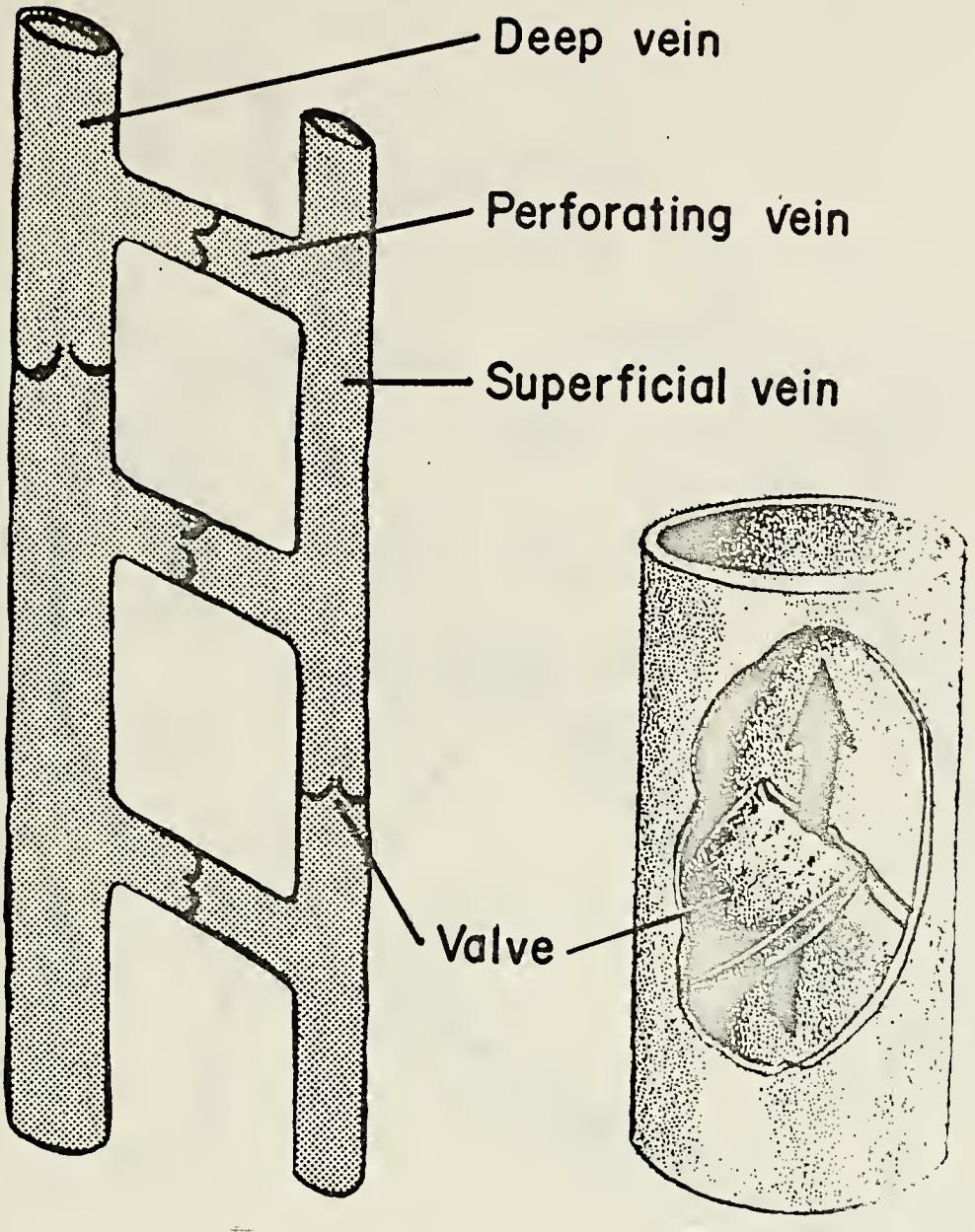



Figure 5. The Venous Valves of the Leg.

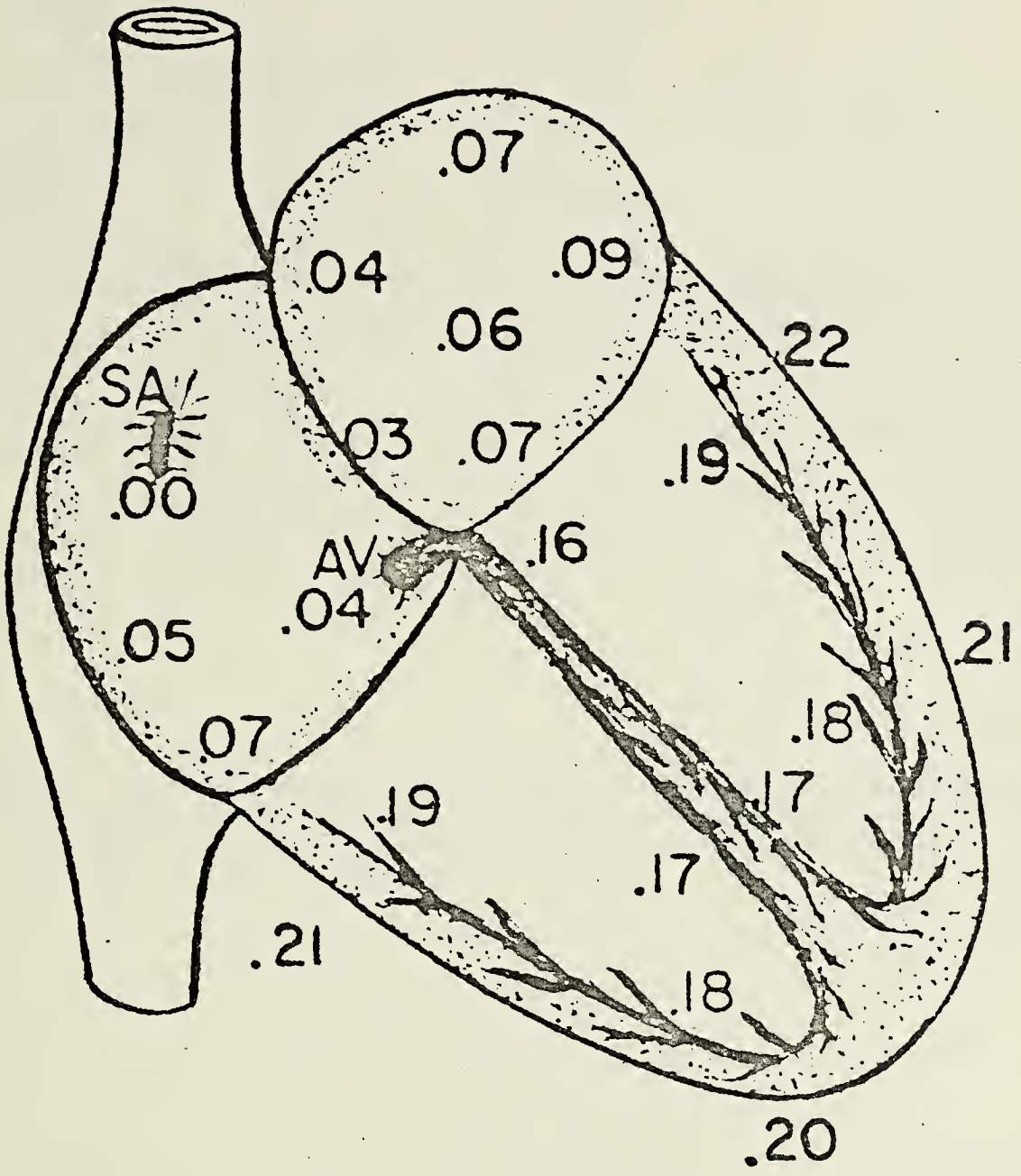



Figure 6. Transmission of the Cardiac Impulse Through the Heart, Showing the Time of Appearance (in fractions of a second) of the Impulse in Different Parts of the Heart.

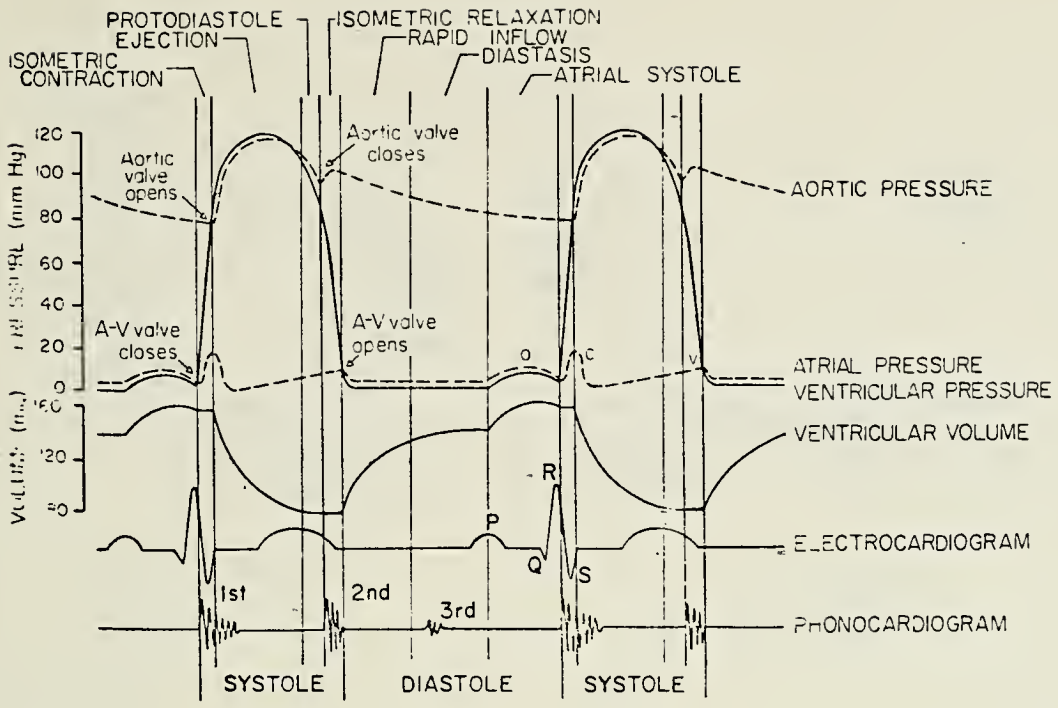



Figure 7. The Events of the Cardiac Cycle, Showing Changes in Left Atrial Pressure, Left Ventricular Pressure, Aortic Pressure, Ventricular Volume, the Electrocardiogram, and the Phonocardiogram.

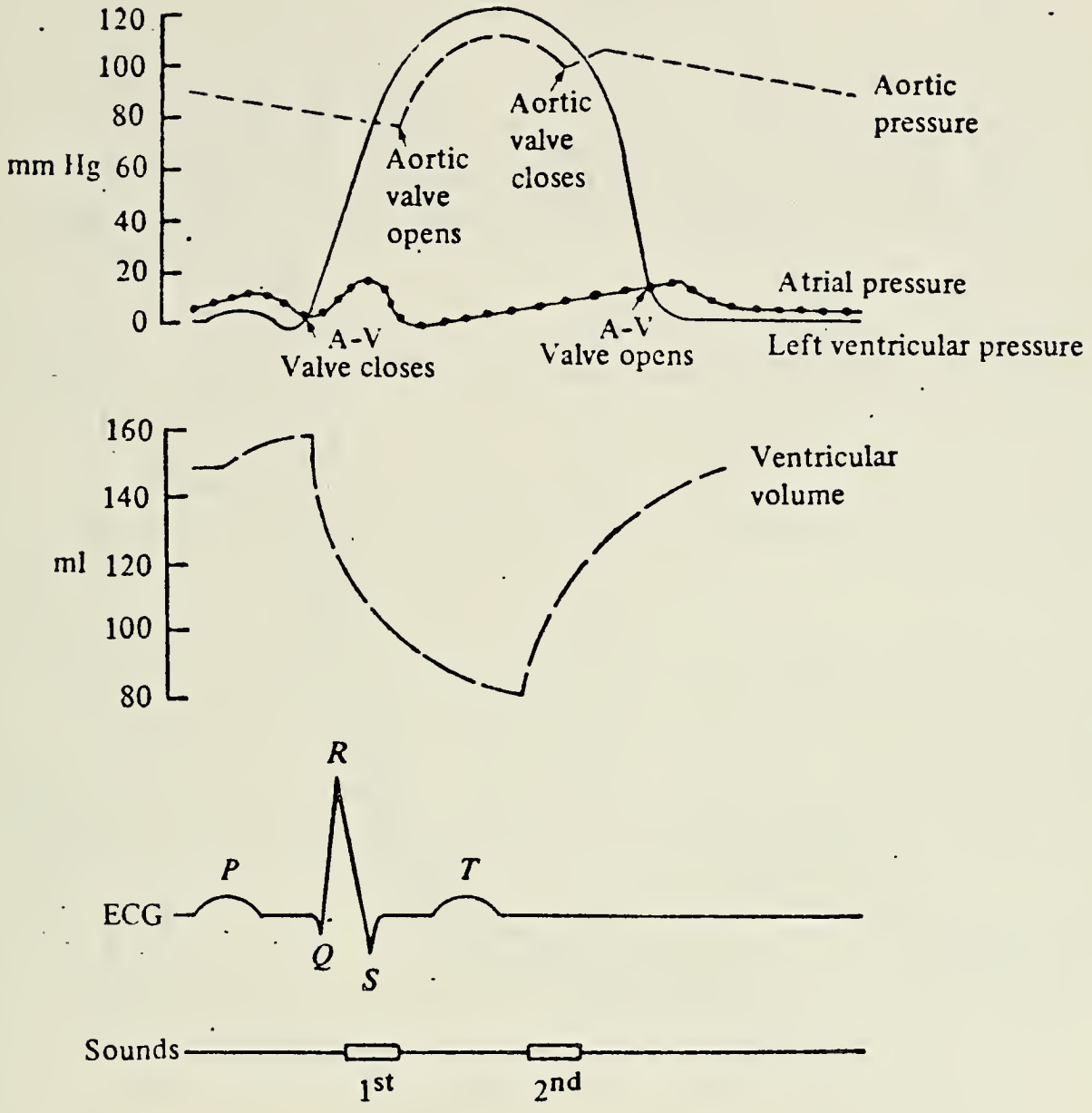



Figure 8. Comparison of Atrial and Left Ventricular Pressures.

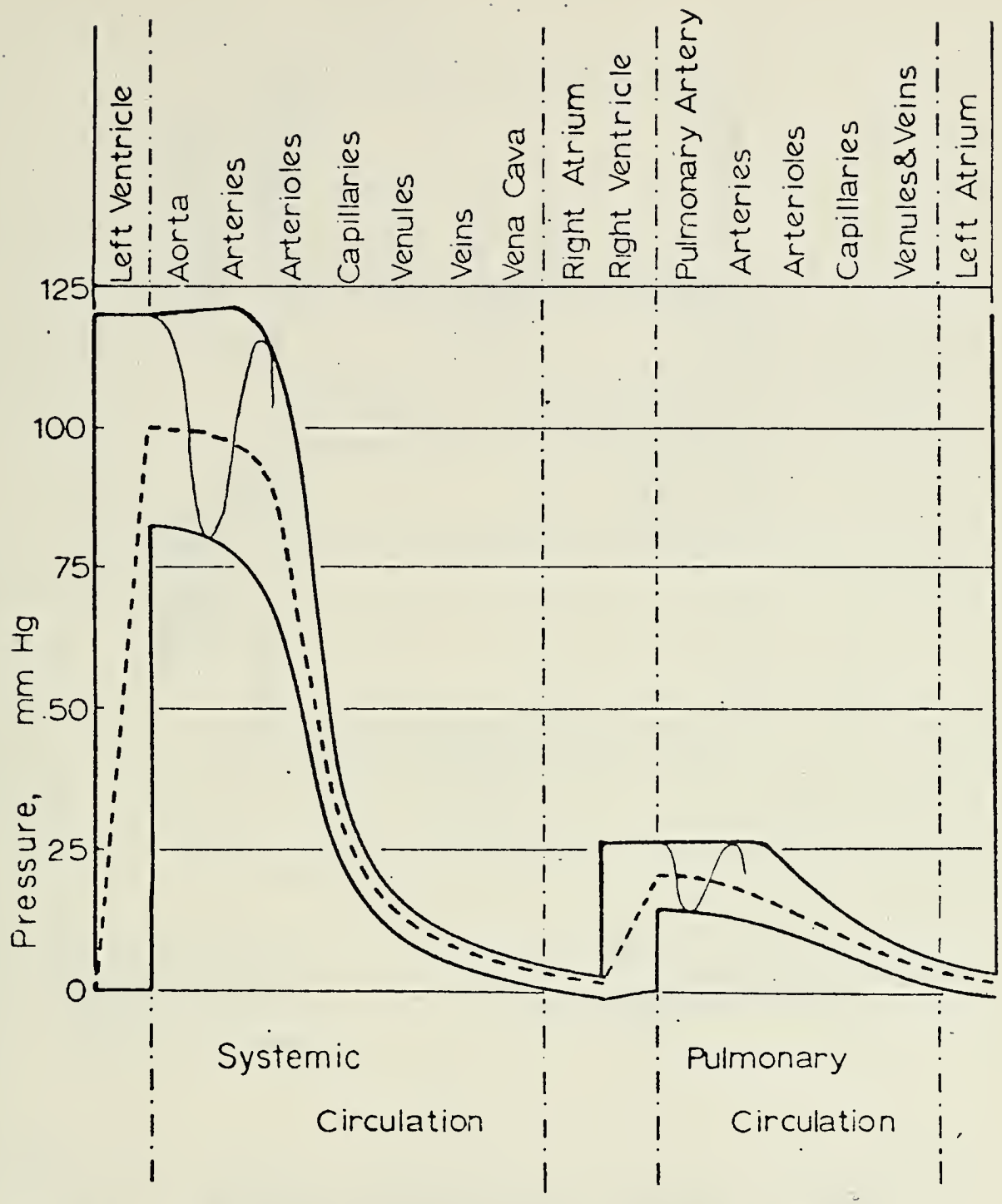
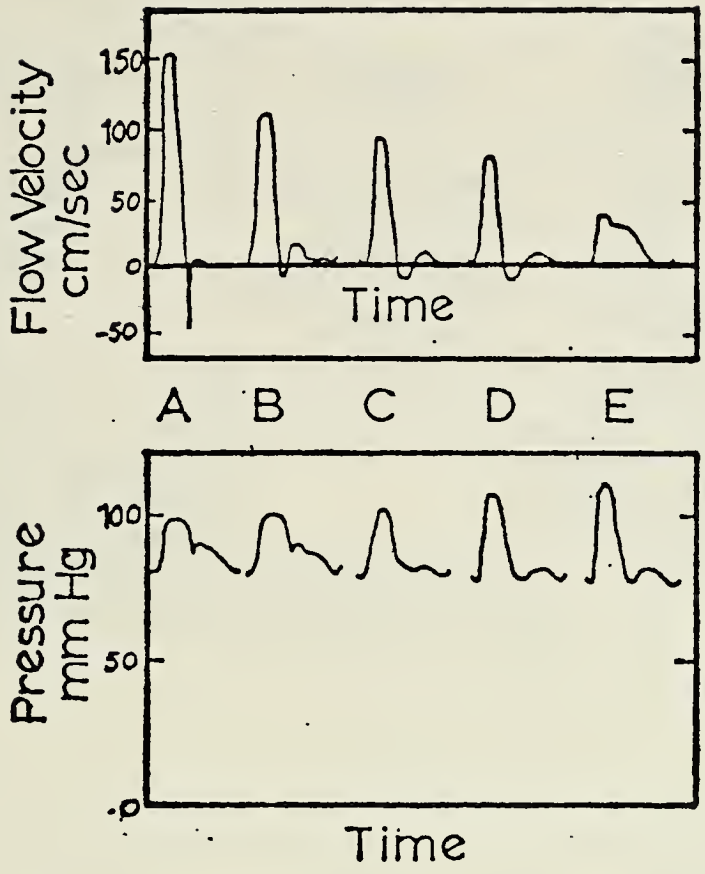



Figure 9. Diagrammatic Representation of the Pressures and Pressure Variations in Various Parts of the Circulation of Man.




A. Ascending aorta
B. Thoracic artery
C. Abdominal artery
D. Femoral artery
E. Saphenous artery

Figure 10. Profiles of the Flow Velocity and Pressure Pulses in the Peripheral Arteries (after McDonald, 1960b).

| Structure | Diameter (cm) | Blood velocity (cm/sec) | Tube Reynolds† number |
|---|---|---|---|
| Ascending aorta | 2·0[1]–3·2[2] | 63±[1] | 3600–5800 |
| Descending aorta | 1·6[1]–2·0[2] | 27±[1] | 1200–1500 |
| Large arteries | 0·2[1]–0·6[1] | 20–50±[1] | 110–850 |
| Capillaries | 0·0005[3]–0·001[3] | 0·05–0·1§[3] | 0·0007–0·003 |
| Large veins | 0·5[3]–1·0[3] | 15–20§[5] | 210–570 |
| Venae cavae | 2·0[4] | 11–16§[4] | 630–900 |

[1] Spencer *et al.* (1963).
[2] Peterson *et al.* (1960).
[3] Maggio (1965).
[5] Brecher (1956).
[4] Helps *et al.* (1954).
† Assuming viscosity of blood is 0·035 poise.
± Mean peak value.
§ Mean velocity over indefinite period of time.

Figure 11. Systemic Circulation of Man (Vessel Size, Blood Velocity, Reynolds Number).

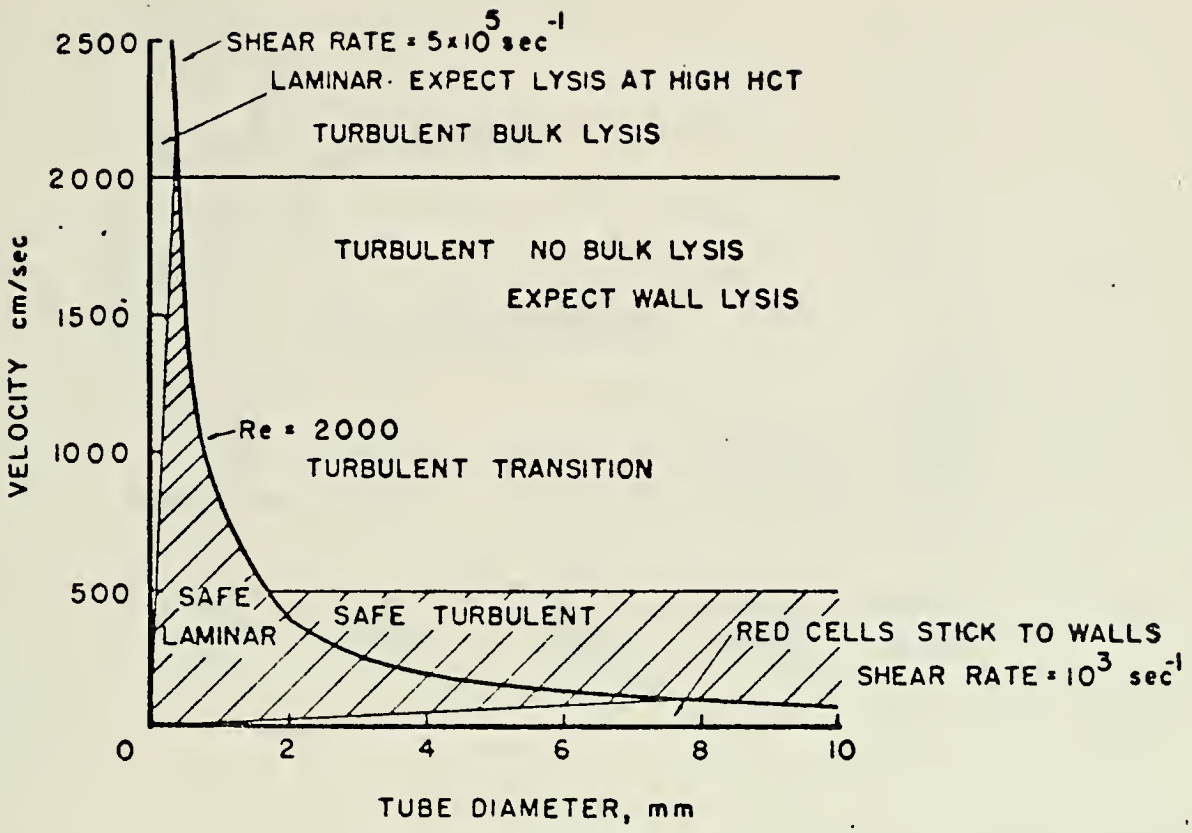



Figure 12. Domains of Fully Developed Flow of Blood in a Tube.

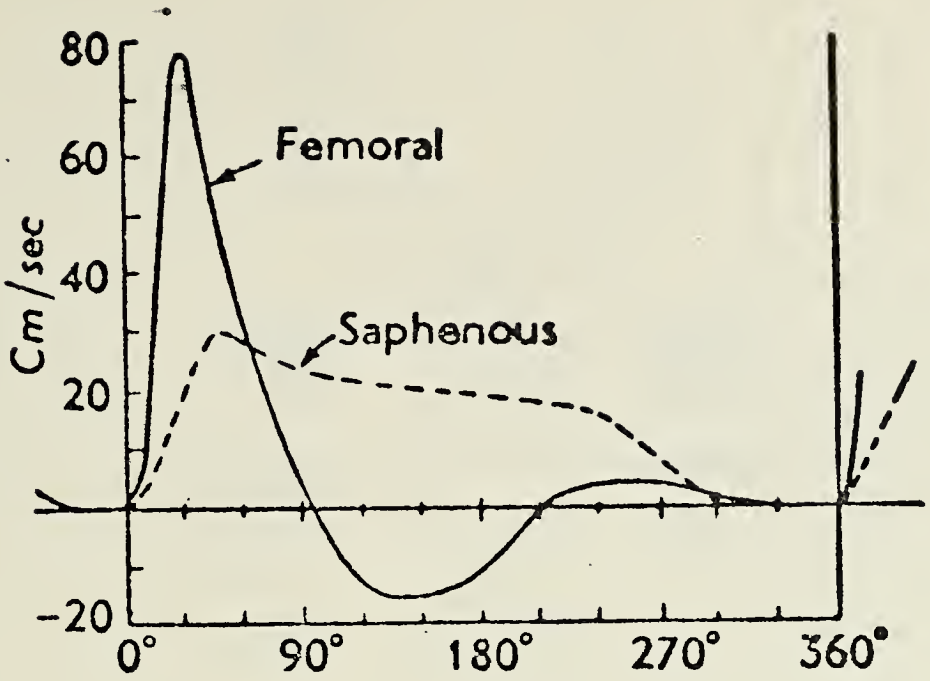



Figure 13. Flow Curves in the Femoral and Saphenous Arteries of the Dog Recorded Simultaneously by High-speed Cinematography.

The flow pattern in the saphenous artery is very different from that in the femoral artery and is attributed to a marked reduction in the oscillatory flow components in the small vessel. This is in part due to damping caused by viscosity but is mainly determined by the large increase in the input impedance especially of the lower frequencies.

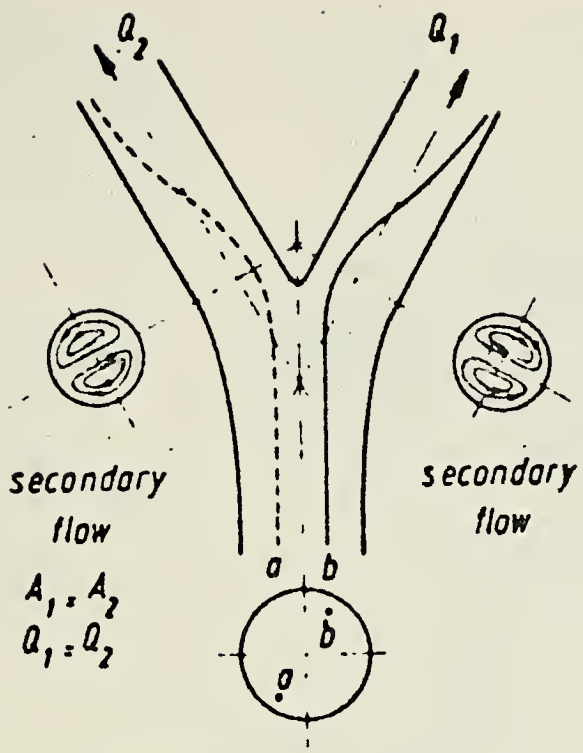
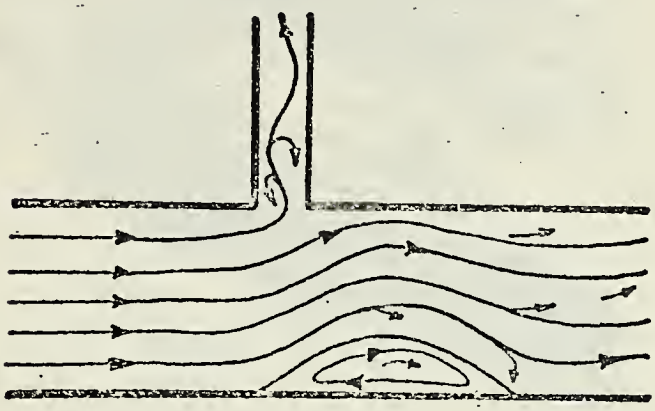



Figure 14. Secondary Flow (Re = 400).

SUDDEN ENLARGEMENT

OUTLET FLOW



INLET FLOW

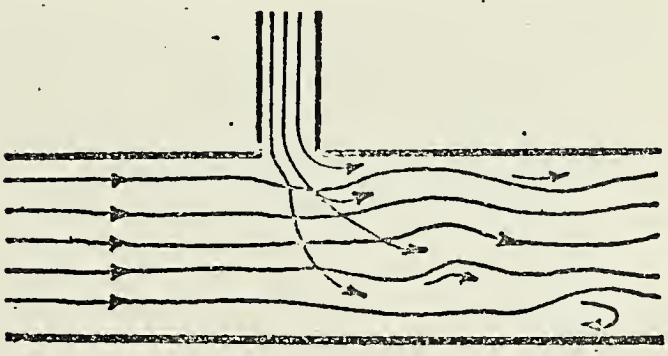


Figure 15. Geometries Conducive to Turbulent Flow.

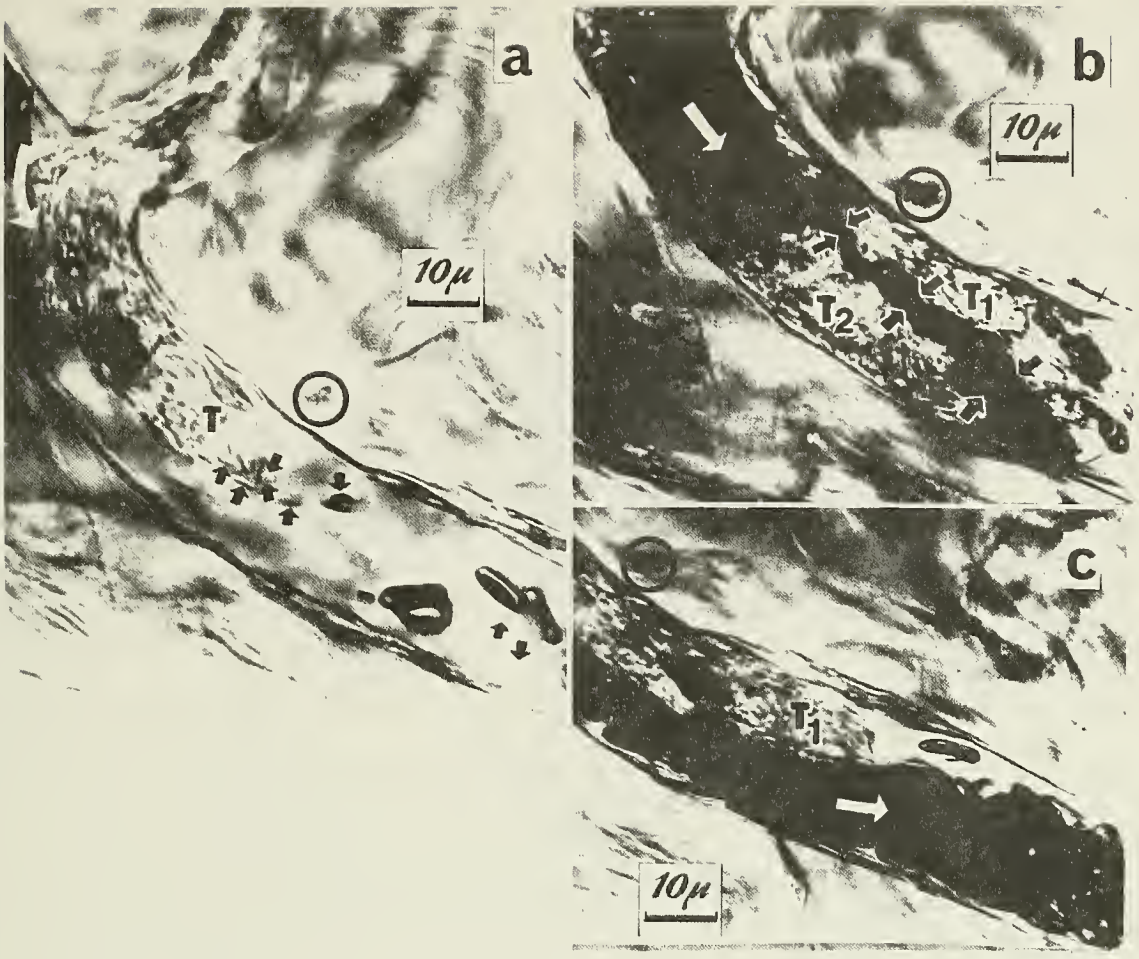



Figure 16. Dynamics of Thromboembolism as Observed by Vital Microscopy in Rabbit Mesentery. Local tissue injury induced formation of platelet thrombus in a small venule. This thrombus (T) was fed into a somewhat larger venule by circulatory force (a). New platelets marked by black arrows were added continuously. In the larger venule, the embolus completely blocked flow of blood for a few minutes, but was then penetrated by red cells being pushed through it, forming an intra-thrombotic channel (marked by black arrows)(b). Lower portion of the thrombus $T_2$ was then released and carried away as an embolus (C), thereby restoring circulatory continuity. Other portion ($T_1$) remained in the vessel as a thrombus.

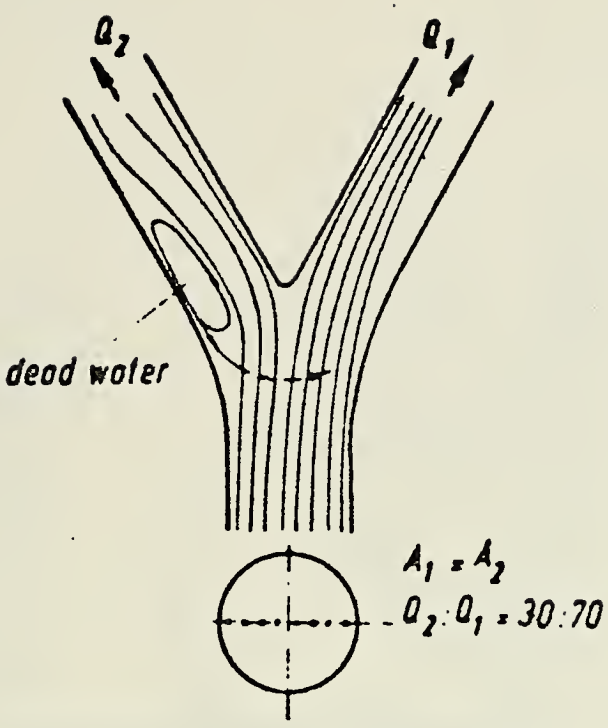



Figure 17. Dead Water and Back Flow (Pe = 1100).

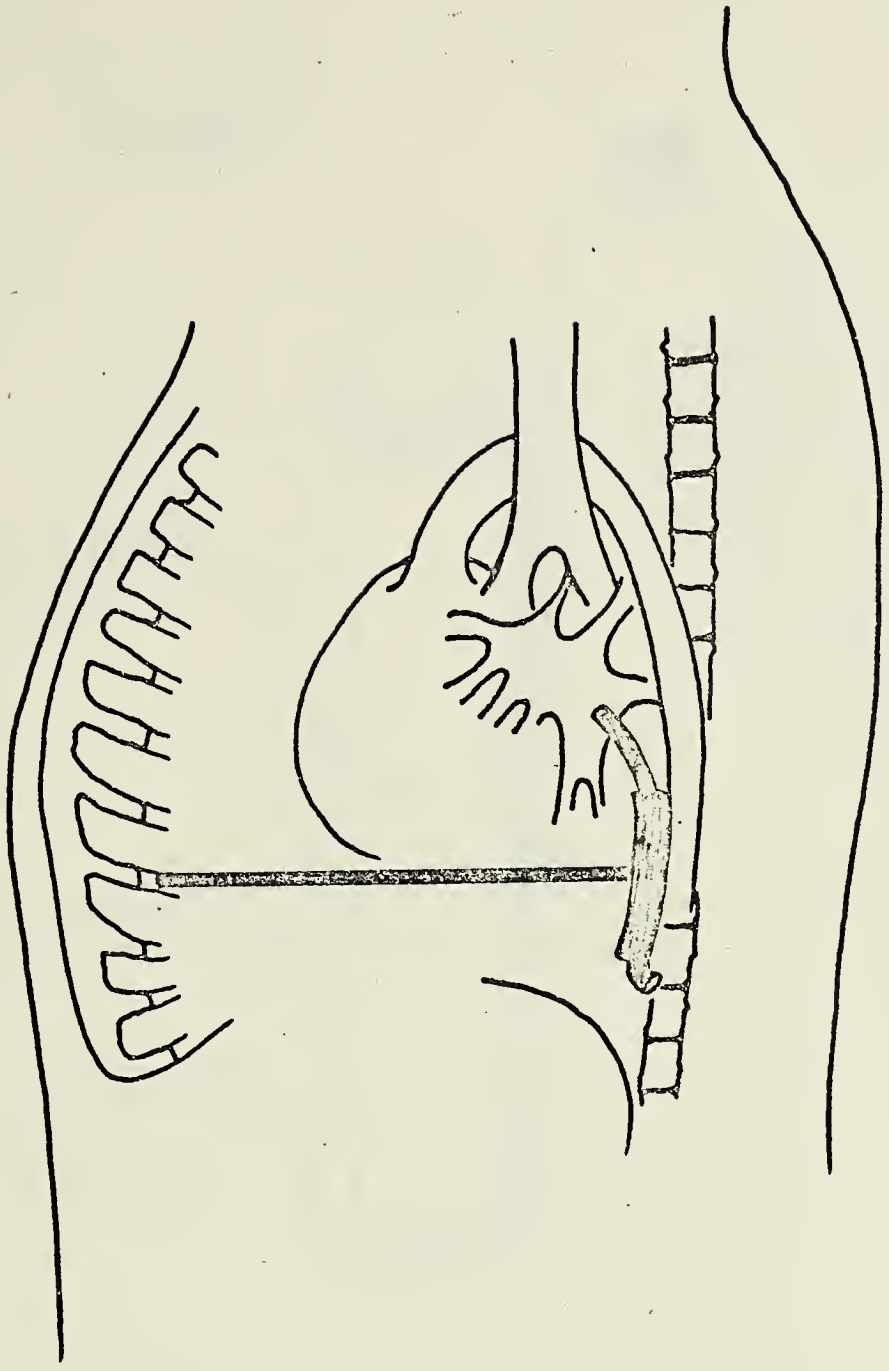


Figure 18. Left Lateral View of Permanent Implanted Assist Device, Shown in Left Atrial to Aorta Bypass Position.

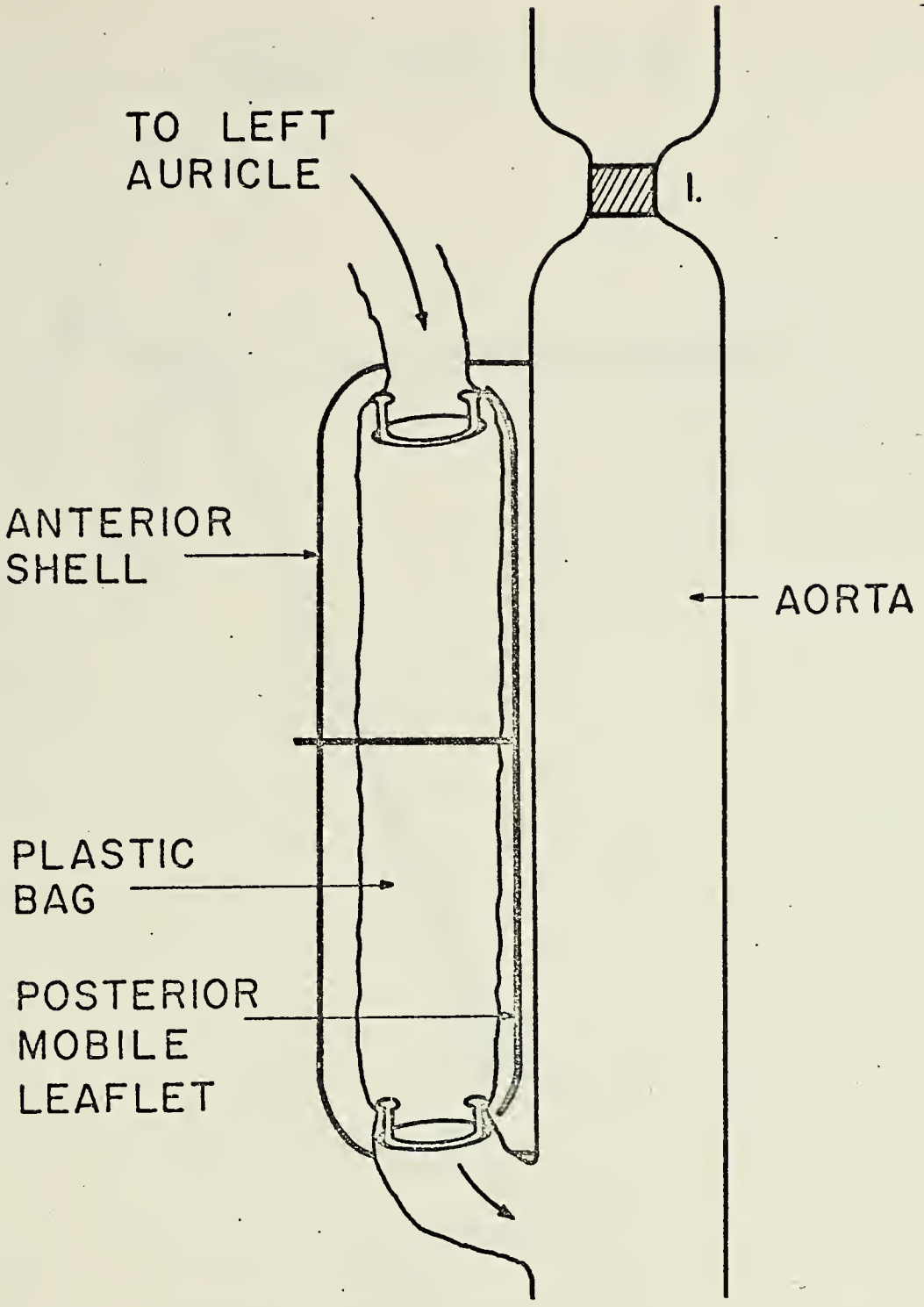



Figure 19. Left Lateral View of Permanent Implanted Assist Device, Showing Lens Valve at Entrance and Exit, Posterior Mobile Leaflet which is Drawn Anteriorly by Chest Wall Motion via Pulley Attachment to Anterior Rib Cage, thence Laterally to Left Lateral Rib Cage, or Alternately by Semi-Rigid Rod Attachment to Anterior Rib Cage. Partial Banding of Aorta above Device.

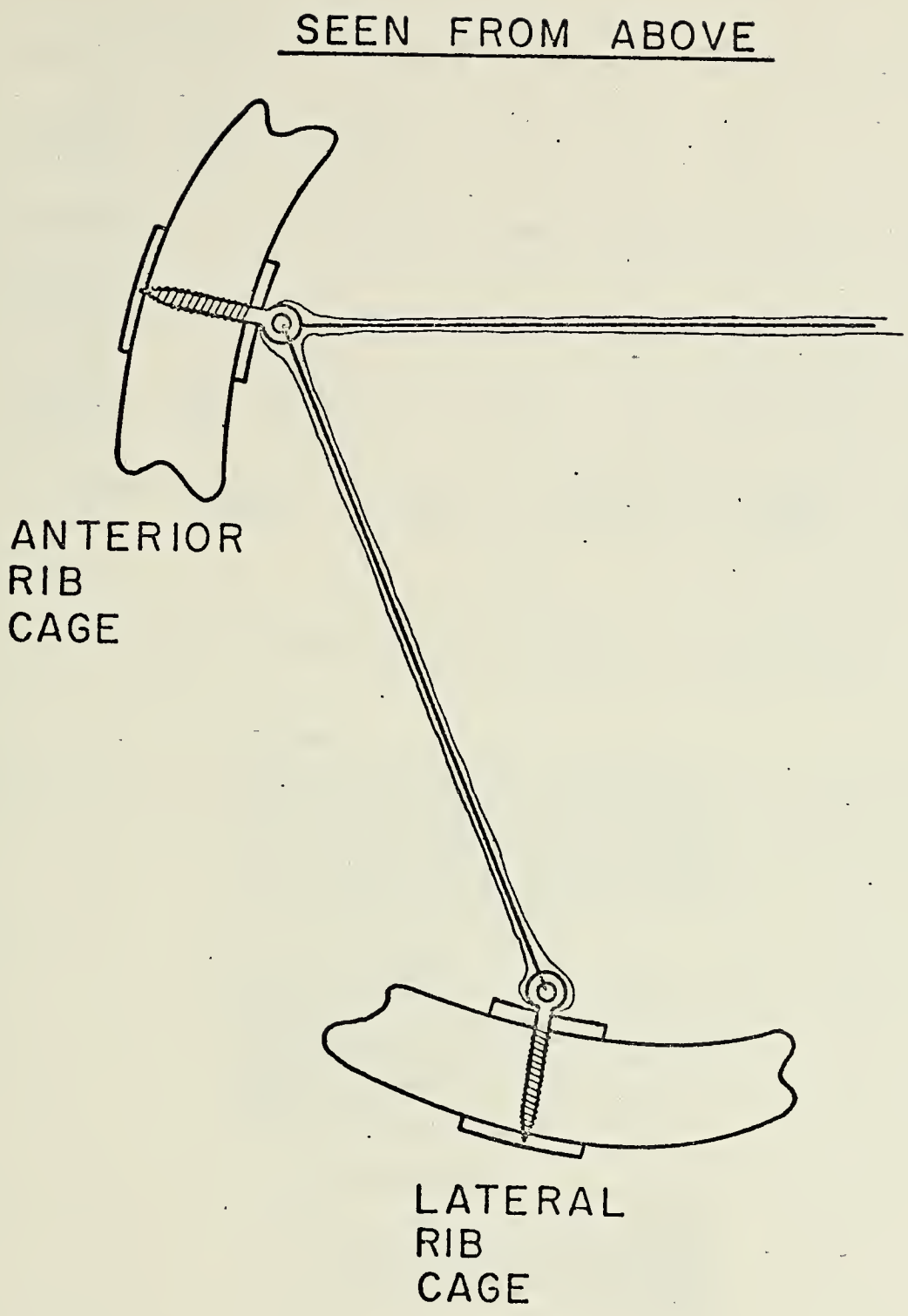
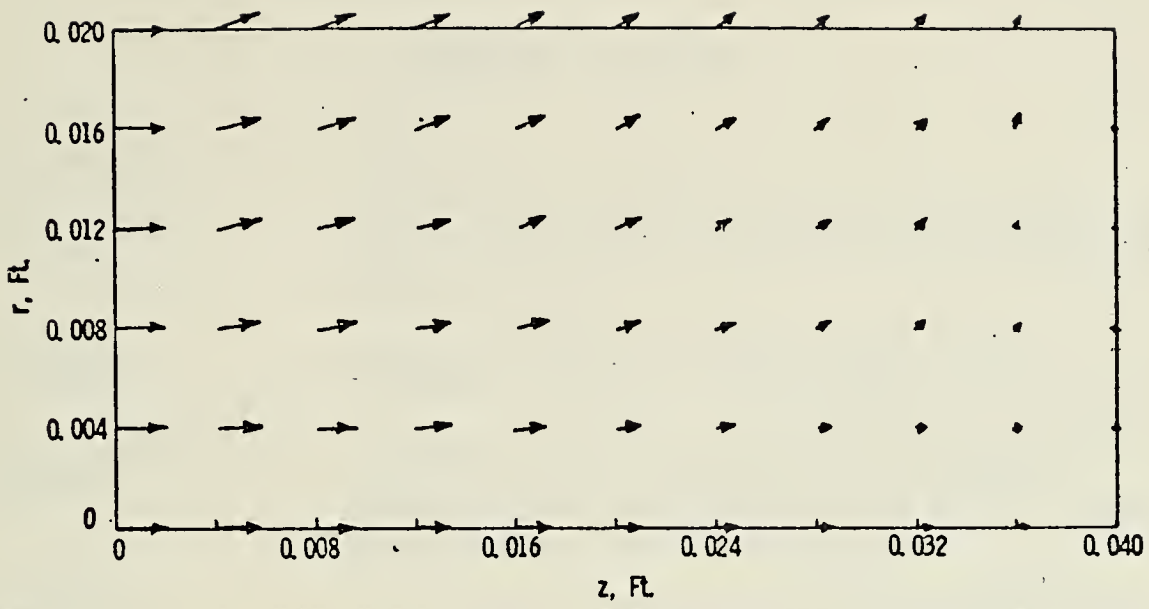
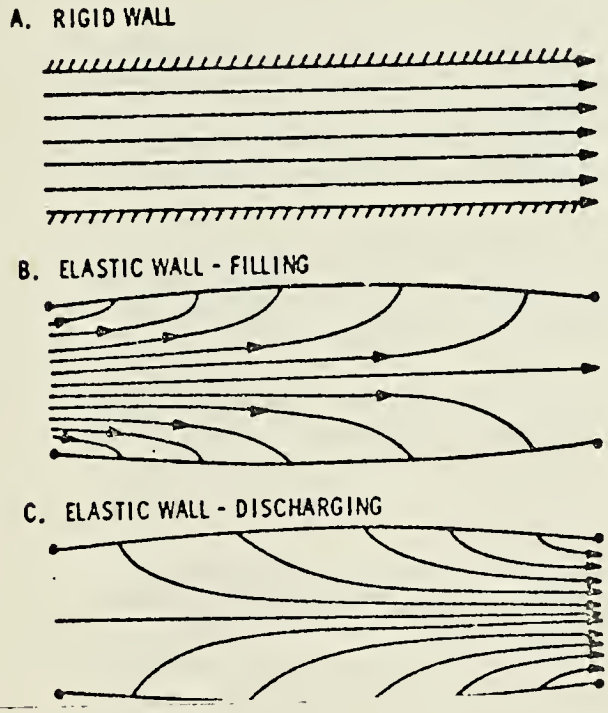



Figure 20. Attachment of Pulley System to Operate Permanently Implanted Assist Device off Thoracic Cage Motion.




Velocity Vectors in a Viscoelastic Blood Vessel




Streamlines in a Viscoelastic Blood Vessel

Figure 21

## GLOSSARY OF TERMS

| | |
|---|---|
| ARTERIOLE | a very small artery |
| ATHEROMA | focal deposits of lipid-containing material; a form of arteriosclerosis with thrombotic complications |
| ATRIUM | the first cavity of the heart which receives blood from the veins |
| ATRIOVENTRICULAR VALVES | tricuspid valves, separating the atria from the ventricles, supported by chordae tendinae and papillary muscle to withstand back pressure during systole |
| BASOPHIL | a cell which stains with basic dyes and increases in number as a degenerative condition related to severe anemia, leukemia and malaria |
| BIFURCATION | division into two branches |
| BINGHAM CONCEPTION | supposition that when pressure applied to the ends of a tube (containing blood) is small, there is a plug flow and an unsheathed core of blood slides within a slippage zone provided by wall effect |
| BOLUS FLOW | in capillary blood flow the red blood cells are commonly observed to move along in trains with cells separated from one another by segments (boluses) of plasma |
| CASSON FLUID | one which possesses finite yield stress and shear dependent viscosity when subjected to periodic pressure gradient in a long tube |
| CORPUSCLE | a protoplasmic cell; floating freely in a fluid, or embedded in a matrix |
| DIASTOLE | the rhythmic period of relaxation and dilatation of a chamber of the heart during which it fills with blood |
| DISCOIDAL | disc-like; flat and circular |
| ENDOCARDIUM | the membrane which lines the inner surface of the heart |

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| ENDOTHELIUM | a squamous (nucleated) epithelium (cellular lining) which lines serous cavities; i.e., the heart, blood vessels |
| EOSINOPHIL | a leucocyte with a two-lobed nucleus and containing cytoplasm filled with granules which stain red under eosin; they significantly increase in number during allergic reaction |
| ERYTHROCYTES | red blood corpuscles |
| FAHREOUS-LINDQVIST EFFECT | phenomenon in microcirculation (where vessel size is of same order as cell size), in which hematocrit, apparent viscosity and flow resistance decrease as the tube diameter is reduced |
| FEMORAL | pertaining to the femur or thigh |
| FIBRIN | an insoluble protein found in blood after coagulation, readily digested in gastric juices |
| FIBROBLASTS | a connective tissue cell which is the catalyst in the conversion of aggregated platelets to full-blown blood clots |
| HEMATOCRIT | the suspension volume fraction occupied by the red blood cells |
| HEMOGLOBIN | the red respiratory pigment of blood of vertebrates |
| HEMOLYSIS | the dissolution of red blood corpuscles with liberation of their hemoglobin |
| HEPARINIZATION | a process by which a substance (heparin) found in the liver and other body tissue is used to render blood non-clotting |
| HYDRODYNAMICS | the principles of dynamics as applied to water and other fluids |
| HYPERTENSION | constriction of arterioles, synonomous with excessive tension and high blood pressure |
| HYPOTHALMUS | the basal part of the diencephalon, believed to contain vital autonomic nervous centers and fiber tracts |
| INTRATHORACIC | within that part of the body between the neck and abdomen, containing the heart, lungs, esophagus, etc. |

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| KINEMATICS | the science which treats of motions considered in themselves, or apart from their causes |
| LAMINAR | arranged in, consisting of, or like laminae (layers) |
| LAMINAR FLOW | streamline flow in a viscous fluid near solid boundaries |
| LEUCOCYTE | a white or colorless blood corpuscle which is amoeboid in nature |
| LIPID MATRIX | a structure composed of fats and other esters, characterized by solubility in fat solvents and insolubility in water |
| LYMPHOCYTE | a white or colorless amoeboid blood cell derived from lymphatic tissue |
| LYSIS | see hemolysis |
| MICROCIRCULATION | microvascular structure and function which is responsible (by capillary perfusion) for the state and function of every single tissue cell and is adjustable to local tissue demand |
| MICROPIPETTES | tubes of very narrow diameter, used to simulate such vessels as the capillaries and smaller venules and arterioles |
| MITRAL | pertaining to or resembling a miter or valve; the cardiac valve guarding the opening between the left auricle and left ventricle and preventing the return of blood to the auricle |
| MONOCYTES | the group of white blood corpuscles including large mononuclear and transition cells |
| MYOCARDIAL | pertaining to the muscular walls of the heart |
| MYOCARDIAL SHORTENING | the shrinkage (contraction) of the cardiac wall muscles, as in the systole |
| NEUTROPHILS | white blood corpuscles (whose granules stain only with neutral stains) which are phagocytic, especially for bacteria |
| NEWTONIAN FLUID | one whose dynamic viscosity ($\mu$) is a property of the fluid itself, and independent of the fluid motion |

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| OCCLUSIVE | tending to shut in or out by closing a passage |
| OSMOTIC | diffusion process through a semipermeable membrane |
| PAPILLARY | a small nipple-like eminence |
| PERICARDIUM | the cavity and membrane enveloping the heart |
| PERISTALTIC | designating or pertaining to the peculiar wormlike wave motion of the intestines and other hollow muscular structures produced by the successive contraction of the muscular fibers of their walls, forcing their contents onward |
| PURKINJE FIBERS | those fibers originating in the atrioventricular node and terminating in the wall between the right and left ventricles transmitting impulses approximately 6 times as rapidly as normal heart muscle |
| PLASMA | the fluid portion of the blood, composed of a mixture of many proteins in a crystalloid solution, and corresponding closely to the interstitial fluid of the body |
| PLATELETS | any of certain colorless discs readily disintegrated, occurring in the blood of animals and important in the blood clotting process |
| PLUG FLOW | analgous to the Bingham plastic, in that three-dimensional cell aggregates form a plug which slides within an annulus of plasma |
| POLYMERS | any of two or more polymeric compounds; specifically one of higher molecular weight |
| POLYMORPHONECLEAR | amoeboid leucocytes with multipartite nuclei connected by fine threads of chromatin |
| PULSATILE | throbbing, pulsating, moving rhythmically |
| REFRACTORY | period after excitation during which repeated stimulus fails to induce a response |
| RHEOLOGICAL | pertaining to science of deformation and flow of matter |

ROULEAUX — formations like piles of coins into which red blood corpuscles tend to aggregate

SAPHENOUS — designating, pertaining to, or in the region of the two principle superficial veins of the leg, the long one passing up the medial side of the leg, the short one passing behind the outer maleolus and up the back of the leg.

SEMILUNAR — pertaining to any of the crescent shaped flaps which are forced apart by pressure in the ventricles during systole and pushed together by pressure in the arteries during diastole, thus preventing regurgitation of blood into the ventricles

SHEAR RATE — the velocity gradient from vessel periphery to center ($\frac{\Delta V}{\Delta r}$ $\frac{cm/sec}{cm}$) in $sec^{-1}$. For a given hematocrit, the higher the shear rate, the lower the viscosity

SINO-ATRIAL NODE — that node within the right atrium of the heart which initiates the cardiac impulse (pacemaker)

SLIPPAGE ZONE — that area (between the vessel wall and the unsheathed core of blood) where the shear stress is concentrated

SULCUS — a groove; specifically those of the heart, tongue, bones, cornea, etc.

SYSTALIC EJECTION — that phase of the heart cycle during which blood is expelled forcibly from the ventricles (into the pulmonary artery or the aorta)

THROMBOCYTES — blood platelets; nucleated spindle-shaped cells concerned with clotting of blood

THROMBUS — a coagulum of blood elements or a growth of cells as tumor cells formed in the heart, a blood vessel, or a lymphatic during life

VENOUS PLEXUS — the network of veins underlying the skin which serves as a radiator in the body's temperature-control function

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| VENULE | a small vein, veinlet |
| VISCOSITY | that property of a body in virtue of which, when flow occurs inside it, forces arise in such a direction as to oppose the flow |
| WINDKESSEL MODEL | a mechanical device (model) used to simulate the varying elasticity of the arterial system and its resulting capability for storing potential energy |

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## DOCUMENT CONTROL DATA - R & D

*(Security classification of title, body of abstract and indexing annotation must be entered when the overall report is classified)*

| 1. ORIGINATING ACTIVITY (Corporate author) | 2a. REPORT SECURITY CLASSIFICATION |
|---|---|
| Naval Postgraduate School<br>Monterey, California 93940 | Unclassified |
| | 2b. GROUP |

3. REPORT TITLE

HYDRODYNAMICS OF THE HUMAN CIRCULATORY SYSTEM: A REVIEW

4. DESCRIPTIVE NOTES (Type of report and inclusive dates)
Master's Thesis; September 1972

5. AUTHOR(S) (First name, middle initial, last name)

Thomas Blake Humphreys; Lieutenant, United States Navy

| 6. REPORT DATE | 7a. TOTAL NO. OF PAGES | 7b. NO. OF REFS |
|---|---|---|
| September 1972 | 100 | 61 |

| 8a. CONTRACT OR GRANT NO. | 9a. ORIGINATOR'S REPORT NUMBER(S) |
|---|---|
| b. PROJECT NO. | |
| c. | 9b. OTHER REPORT NO(S) (Any other numbers that may be assigned this report) |
| d. | |

10. DISTRIBUTION STATEMENT

Approved for public release; distribution unlimited.

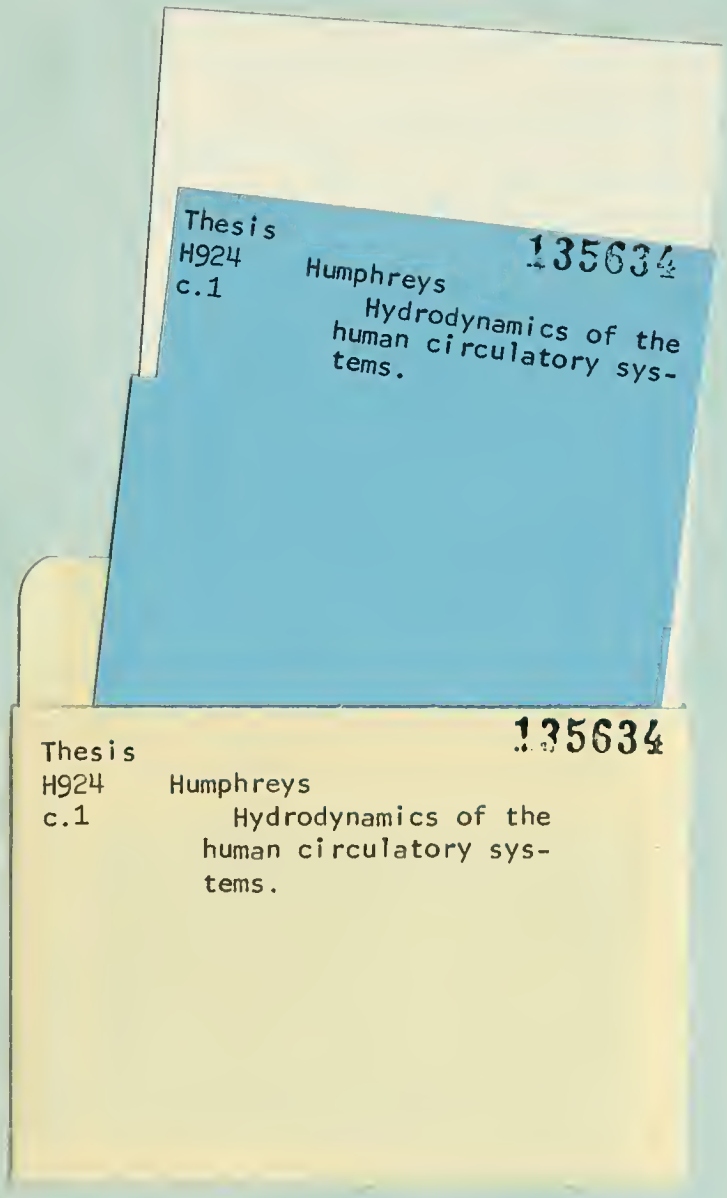
| 11. SUPPLEMENTARY NOTES | 12. SPONSORING MILITARY ACTIVITY |
|---|---|
| | Naval Postgraduate School<br>Monterey, California 93940 |

13. ABSTRACT

The results of a literature survey of blood flow are used to determine the extent to which the principles of hydrodynamics are being employed in the study of the human circulatory system. Blood flow is categorized by such parameters as Reynold's number, vessel diameter and geometry, and transport mechanism with a view toward understanding the rheological behavior of blood in various flow regimes.

This study provides an engineering explanation of the hydrodynamic origin of such physiological disorders as arteriosclerosis, atheroma, and thromboembolism. Moreover, it provides an appreciation for the engineering aspects in the design of cardiac assist and cardiac replacement devices.

| 14. KEY WORDS | LINK A | | LINK B | | LINK C | |
|---|---|---|---|---|---|---|
| | ROLE | WT | ROLE | WT | ROLE | WT |
| HYDRODYNAMICS | | | | | | |
| CIRCULATION | | | | | | |
| BLOOD FLOW | | | | | | |
| HEMODYNAMICS | | | | | | |
| HEMORHEOLOGY | | | | | | |



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Hydrodynamics of the human circulatory systems.
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